VOLUME VI • NUMBER 4 • NOVEMBER 1961

SERIALS
APR 5 1965

ANNALS OF PHYSICAL MEDICINE

The Official Organ of the British Association of Physical Medicine

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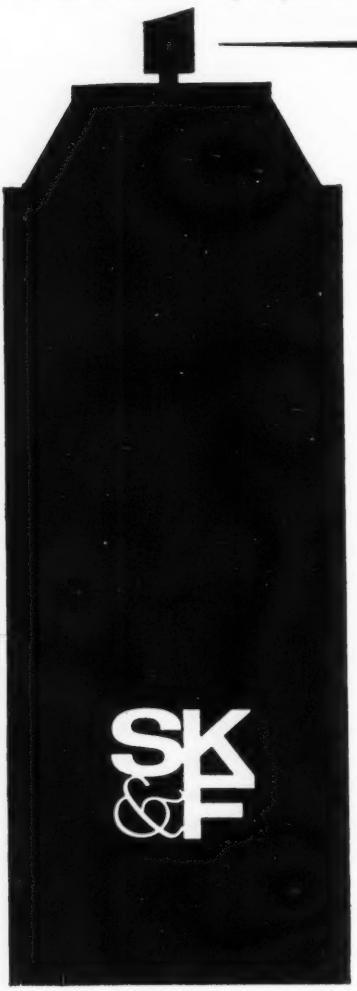

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ANNALS OF PHYSICAL MEDICINE

The Official Organ of the
BRITISH ASSOCIATION OF PHYSICAL MEDICINE

Vol. VI November 1961 No. 4

ORIGINAL PAPER

THE EFFECT OF TREATMENT ON MEDIAN NERVE CONDUCTION IN PATIENTS WITH THE CARPAL TUNNEL SYNDROME

By H. V. GOODMAN and R. W. GILLIATT

From the Institute of Clinical Research, Middlesex Hospital Medical School, London

SLOWING of nerve conduction in patients with the carpal tunnel syndrome was first reported by Simpson in 1956. When the median nerve was stimulated at the wrist and the muscle response recorded from the abductor pollicis brevis, it was found that the latency of the muscle action potential was significantly increased, the abnormality being reversed by surgical division of the flexor retinaculum.

In the same year that Simpson published his investigation of motor latency, Dawson (1956) described a method of recording action potentials from the median and ulnar nerves at the wrist when the sensory fibres in the fingers were stimulated. Small ring electrodes were wrapped round the fingers to stimulate digital nerve fibres, and nerve action potentials were recorded by means of surface electrodes placed over the median or ulnar nerve trunks just above the wrist. Using this technique, it was subsequently shown by Gilliatt and Sears (1958) that median nerve action potentials might be delayed or lost altogether in patients with the carpal tunnel syndrome.

These observations suggested that the examination of nerve conduction could provide an accurate and objective method of assessing the degree of nerve damage in patients with the carpal tunnel syndrome. It was therefore decided to study a group of patients both before and after surgical division of the flexor retinaculum in order to discover whether abnormalities of nerve conduction would resolve completely after surgery, and, if so, after what interval. For this purpose it was

* Based on a paper read at the Annual Meeting of the British Association of Physical Medicine, London, April, 1960.

important to ensure that the flexor retinaculum was completely divided in each case. Accordingly, all patients in this group were operated on by a standard technique which will be described in a later section.

In view of the symptomatic improvement observed by Heathfield (1957) in patients with the carpal tunnel syndrome treated by splinting of the wrist, it was decided to investigate a second group of patients treated in this way, and to study nerve conduction before and during the period of splinting. Later, a third group of patients was studied who received injections of prednisolone into the carpal tunnel deep to the median nerve. The investigation of this third group was carried out jointly with Dr. J. B. Foster, and will be reported separately (Goodman and Foster, to be published).

METHODS

Patients were examined lying on a couch with the arm outstretched on a pillow. When the hands were cold they were immersed in hot water for five minutes before the test, but no other method of temperature control was attempted. The abductor pollicis brevis was identified by inspection and palpation during abduction of the thumb, and a coaxial needle electrode inserted into the belly of the muscle. In wasted muscles the needle was placed where surviving muscle fibres could be detected by palpation over the shaft of the first metacarpal. The median nerve was then stimulated through the skin at a point 2–3 cm. above the distal wrist crease. The stimulating cathode was a small pad electrode 1 cm. in diameter, and the anode was a metal plate 5 cm. by 3·5 cm. placed on the flexor aspect of the forearm (Fig. 1).

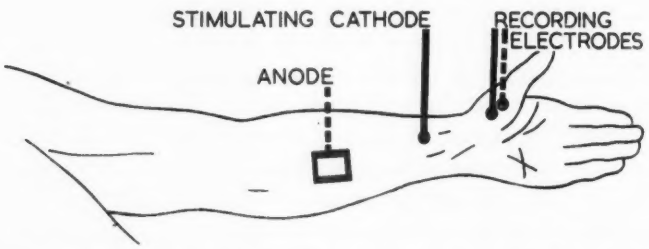

FIG. 1.—Position of stimulating and recording electrodes for estimation of motor latency.

The stimulus, which was delivered through an isolating transformer, was a rectangular pulse of 0·1 msec. duration with a continuously variable voltage up to 150 volts. Skin resistance was lowered by preliminary washing of the skin and rubbing with abrasive jelly. Between 40 and 60 volts were usually required to provide a maximal stimulus for motor fibres in the normal median nerve. This voltage was often inadequate for pathological nerve fibres, and in such cases stimulus intensity was increased in order to produce a muscle response of maximal amplitude.

For recording the muscle response, action potentials were led off from the abductor pollicis brevis, and after suitable amplification displayed on a cathode-ray oscilloscope. A Stanley Cox double-channel electromyograph was used, the second channel providing a 200-cycle-per-second time scale. The stimulus was used to trigger the time-base of the oscilloscope, and the latency of the muscle response measured from the commencement of the trace on photographic records.

Sensory nerve action potentials were recorded using a technique similar to that of Dawson (1956). The stimulating electrodes were small silver strips covered in lint and soaked in saline; they were wrapped round the index finger, the cathode being placed at the base of the finger and the anode at the level of the distal interphalangeal joint. Recording electrodes of the saddle type described by Dawson and Scott (1949) with a fixed inter-electrode distance of 4 cm. were placed over the median nerve trunk with the lower electrode 2–3 cm. above the distal wrist crease. A large pad electrode on the back of the hand or wrist was connected to earth, and skin resistance under the recording electrodes lowered to less than 5 kilohms by means of abrasives in order to reduce stimulus artefact. Even in normal subjects the sensory action potential recorded at the wrist with stimulation of the index finger is of relatively small amplitude, and in order to identify the potential in the presence of background activity it was usually necessary to superimpose 5 or 10 traces on the same piece of film.

In the measurement of motor latency, conduction distance from the stimulating cathode to the recording needle varied in different subjects from 5 to 8 cm. When recording sensory action potentials, conduction distance between the stimulating cathode and the lower recording electrode varied from 11 to 15 cm. in different subjects. No attempt was made to correct for variations in conduction distance in different subjects, but in serial examinations on the same subject electrode positions were adjusted to maintain a constant conduction distance on each occasion.

RESULTS

I. Motor Conduction

ELECTRICAL ABNORMALITIES PRESENT BEFORE TREATMENT

Forty-four patients were examined before and at regular intervals after treatment. In 3 patients the effects of treatment were studied in both hands at different times. All patients were seen initially at the Middlesex Hospital between May, 1957, and April, 1959, follow-up studies continuing in some of them until April, 1960. In order to compare different methods of treatment a simple grading of severity was attempted on the basis of motor conduction time between the wrist and thenar muscles. In the series of 50 control subjects studied by Thomas (1960), whose technique was similar to that used in the present survey, the mean conduction time from just above the wrist to the abductor pollicis brevis was 3·8 msec. (S.D.$\pm$0·5), con-

duction distance varying between 6 and 8 cm. For the present survey it was decided to classify as abnormal all motor latencies of more than 5 msec., a latency of 5–7 msec. being regarded as a mild delay, a latency of over 7 msec. and less than 10 msec. a moderately severe delay, and a latency of more than 10 msec. a severe delay. Illustrative tracings are shown in Fig. 2.

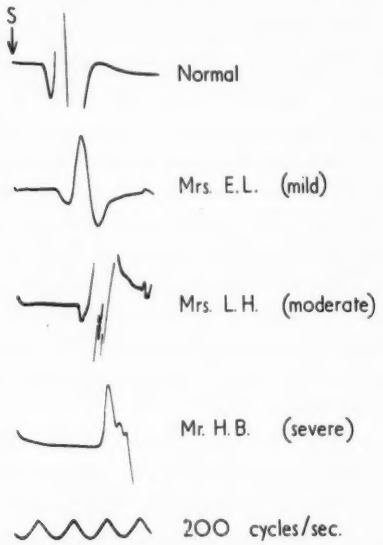

FIG. 2.—Records from abductor pollicis brevis with median nerve stimulation just above wrist. Stimulus (S) at commencement of trace in each case. Top record shows normal response; lower records show mild, moderate, and severe conduction delays.

Twenty patients were treated by surgical division of the flexor retinaculum and 25 patients by splinting of the wrist at night. It was, of course, inevitable that of the severe group most would require operation, but, since two severely affected patients were reluctant to undergo surgery, it was possible to try splinting in these two patients who would otherwise have been recommended for surgery on the grounds of severity alone. For the whole series of patients the conduction findings are shown in Table I, and allotment to the two treatment groups in Table II.

In Table III the presence of wasting and weakness on clinical examination is shown in relation to the conduction delay. From this table it is clear that, although there was a tendency for patients with obvious clinical evidence of wasting and weakness to show severe impairment of nerve conduction, the correlation was not by any means a close one. In general, the electrical abnormalities tended to be more marked than the clinical findings, and 15 patients with normal muscle bulk and power in the thenar muscles showed a conduction delay which was moderately

severe in seven and severe in one. There were six patients in whom the median nerve was inexcitable on percutaneous stimulation at the wrist. In four of them some voluntary activity was still present, and it is possible that a response to nerve

TABLE I

GRADING OF PATIENTS ACCORDING TO CONDUCTION DELAY

(47 hands examined in 44 patients)

	No. of Cases*
Severe delay (more than 10 msec.)	12
Moderate delay (7–10 msec.)	14
Mild delay (5–7 msec.)	15
Normal latency (less than 5 msec.)	6
Total	47

* In this table and subsequent tables "Cases" refers to "Hands".

TABLE II

ALLOTMENT OF CASES TO TREATMENT GROUPS

Treatment	Conduction Delay				Total Cases
	Normal	Mild	Moderate	Severe	
Surgery	3	4	6	10	23*
Splinting	3	12	8	2	25

* Includes 1 case in which splinting had failed.

TABLE III

RELATION OF CLINICAL SIGNS TO CONDUCTION DELAY

	Conduction Delay				Nerve Inexcitable
	None	Mild	Moderate	Severe	
Thenar wasting	1	2	2	3	5
Thenar weakness without wasting	1	6	5	2	1
Clinically normal	4	7	7	1	0

stimulation would have been obtained at a higher stimulus intensity than that available from our stimulator. However, this would have involved stimulation under anaesthesia, as the highest voltage shocks which we were able to deliver came close to the maximum tolerated by a conscious patient.

SURGICAL TREATMENT

Twenty patients were treated by surgical division of the anterior carpal ligament between May, 1957, and August, 1958. In three patients both hands were operated on at different times, so that in all surgery was carried out in 23 hands. Clinical details of these patients are given by Goodman (1960) and only the electrical findings will be described here.

Surgical division of the flexor retinaculum was carried out by Mr. Valentine Logue, to whom we are indebted for the following account of the procedure. The operation was performed using a pneumatic tourniquet round the upper arm so as to ensure a bloodless field. A longitudinal skin incision was used, starting at the distal wrist crease and extending distally for 4 cm. in the palmar crease. Through this incision the whole length of the flexor retinaculum could be displayed and divided under direct vision and the median nerve could be inspected. The tourniquet was then removed and all bleeding arrested before closing the skin incision.

The actual appearance of the median nerve was recorded at operation in all of our cases. No abnormality of calibre of the nerve was seen in 13 cases, although in four of these the nerve appeared to be abnormally vascular when the tourniquet was released. Some broadening or flattening of the nerve was seen in five cases, while a definite constriction under the retinaculum was seen in three others. Only two cases showed a marked swelling proximal to the retinaculum; in both of them the median nerve was electrically inexcitable before operation. In another case, however, the median nerve was also electrically inexcitable preoperatively, but no abnormality was seen on inspection of the nerve at operation.

Division of the retinaculum under direct vision was regarded as essential in this investigation to ensure that slow or incomplete recovery could not be related to incomplete division of the distal fibres of the ligament (a recognized hazard in any "blind" operation carried out through a small incision at a higher level). Two out of 20 patients in this series had been operated on previously by the "blind" method without symptomatic relief, and in both of them direct inspection at the second operation revealed that only the proximal fibres of the retinaculum had been divided (Cases 3 and 16). In one further patient who has not been included in the main surgical series, electrical records were obtained before an unsuccessful "blind" operation, and subsequently further records were obtained before and after re-exploration by Mr. Valentine Logue. The findings in this patient will be described in more detail in a subsequent section.

The clinical results of surgery were satisfactory in all cases, relief of pain being rapid and complete. Wasting was slow to disappear in some of the severely affected patients, and at the end of the follow-up period mild wasting or weakness was still detectable in seven cases and a slight residual sensory disturbance in seven cases also. Further details are described for individual patients by Goodman (1960).

EFFECTS OF OPERATION ON MEDIAN NERVE CONDUCTION

Group I. Severe Conduction Delay.—In the 10 severe cases latency measurements before and after operation are shown in Fig. 3. In all, 68 examinations were carried out in the 10 cases. The shortest period of follow-up was 41 weeks and the longest period 104 weeks. From Fig. 3 it can be seen that the median nerve was initially inexcitable in four patients and that in one of them it remained inexcitable for as long as 29 weeks after operation.

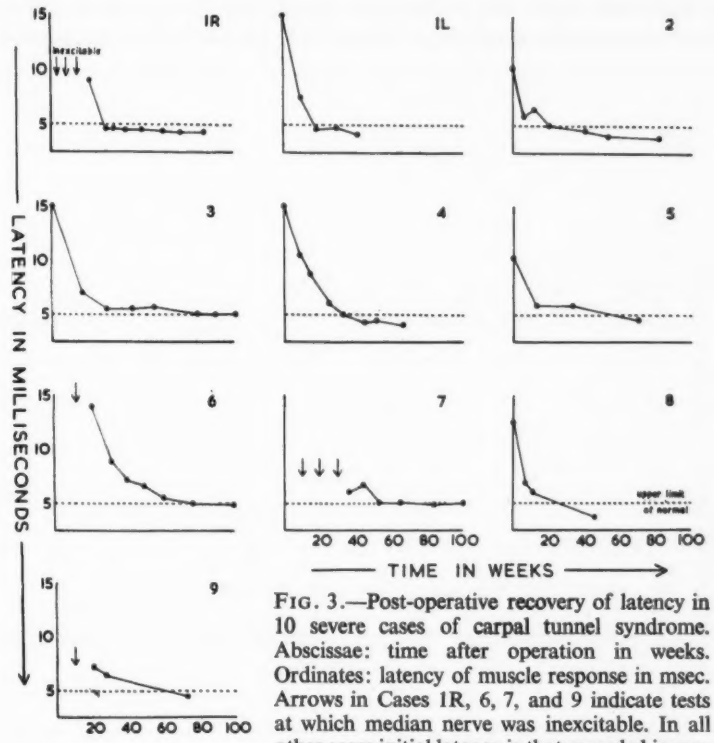

FIG. 3.—Post-operative recovery of latency in 10 severe cases of carpal tunnel syndrome. Abscissae: time after operation in weeks. Ordinates: latency of muscle response in msec. Arrows in Cases 1R, 6, 7, and 9 indicate tests at which median nerve was inexcitable. In all other cases initial latency is that recorded immediately before operation.

It is clear from Fig. 3 that in all cases motor latency returned gradually to within the normal range, although in three of them (Cases 3, 6, and 7) 18 months elapsed before this occurred. The shape of the curves in Fig. 3 suggests that in most cases a constant state had been reached by the end of the follow-up period, so that the final figures for latency may be regarded as representing the best result to be expected after adequate operation and prolonged follow-up in cases of this severity.

For the 10 cases the mean final latency was 4·4 msec., which may be contrasted with the mean figure of 3·8 msec. obtained by Thomas (1960) in control subjects. From this it may be suggested that complete restoration of normal motor conduction should not be expected in those patients with severe preoperative conduction defects.

Group II. Moderately Severe Conduction Delay.—In six cases with moderately severe nerve damage latency measurements are shown in Fig. 4. Forty-one examinations were carried out in the six cases, the shortest period of follow-up being 68 weeks and the longest 90 weeks. From Fig. 4 it can be seen that electrical recovery was more rapid and complete in this group than in the severe cases. In three of the moderately severe cases (Cases 12R, 14, and 15) latencies were still at

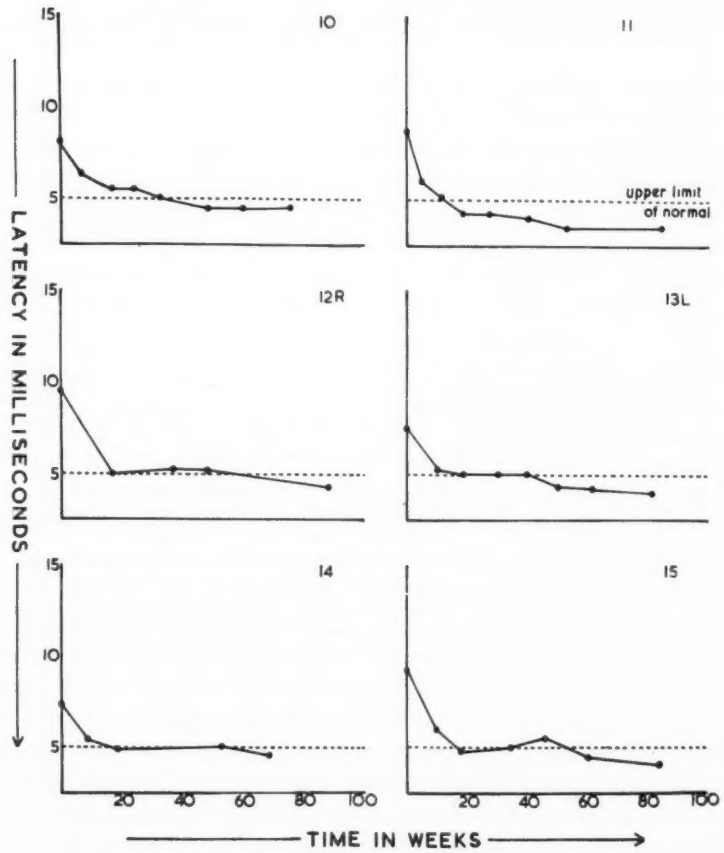

FIG. 4.—Post-operative recovery of latency in 6 patients with moderately severe conduction delays. Display as in Fig. 3.

the upper limit of normal at the end of the first post-operative year, with slow improvement still continuing during the second year, but in the remaining three cases latencies had returned to the normal range at the end of eight months. In all cases a final value for latency was reached which was within the normal range, the mean figures for the six cases being 4·2 msec.

Group III. Mild Conduction Delay.—Four patients were included in this group; 25 examinations were carried out in the four cases and the period of follow-up varied from 36 to 126 weeks. Latency measurements are shown for this group before and after operation in Fig. 5, and it is clear that recovery was more rapid than in the preceding groups, the upper limit of normal being reached within six months in all four cases. The mean final latency for the four cases was 3·9 msec., which is close to the mean figure found by Thomas (1960) in control subjects.

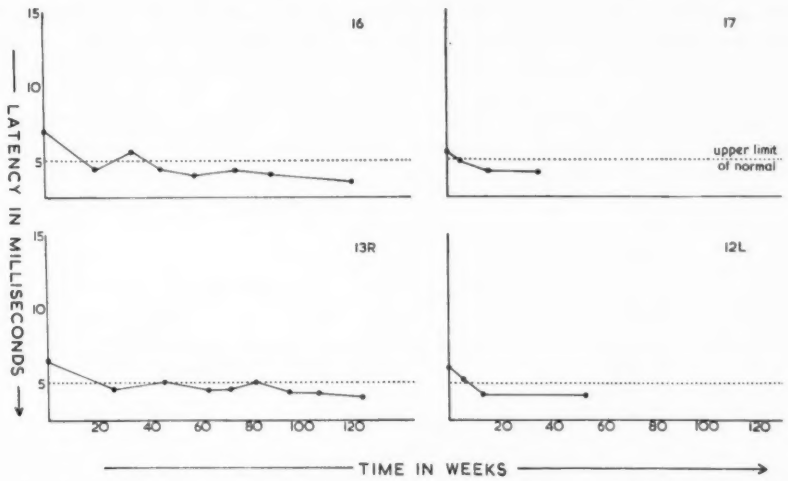

FIG. 5.—Post-operative recovery of latency in 4 patients with mild conduction delays. Display as in Fig. 3.

Group IV. Normal Motor Latency.—There were three cases in which the pre-operative figures for motor latency were within normal limits (less than 5 msec.). These patients were followed up for periods varying between 60 and 93 weeks, a total of 15 examinations being carried out. Latency measurements are given for these patients in Table IV, from which it can be seen that in two cases latency values decreased during the follow-up period and that in the third case there was no change.

Combined Results for Groups I, II, and III.—Results for the three groups with delayed conduction in the pre-operative period are shown graphically in Fig. 6, in which post-operative latencies have been plotted as percentages of the initial

TABLE IV

LATENCY MEASUREMENTS—SURGICAL SERIES: NORMAL MOTOR LATENCIES

Case No.		Pre-operative		Post-operative				
18	Latency (msec.)	4·4	4·4	4·3	3·8	4·0	3·6	3·3
	Time interval (weeks) ..	6	1	7	13	35	59	89
19	Latency (msec.)		4·7	4·7	4·0	3·7	3·7	3·1
	Time interval (weeks) ..		1	14	31	44	58	93
20	Latency (msec.)		4·3	4·3				
	Time interval (weeks) ..		3	60				

(pre-operative) value. The four cases in which the nerve was inexcitable before operation have been excluded from Fig. 6 as an initial figure for latency could not be obtained, but results for the remaining 16 cases are shown and demonstrate clearly the uniform improvement seen in all three groups after surgery.

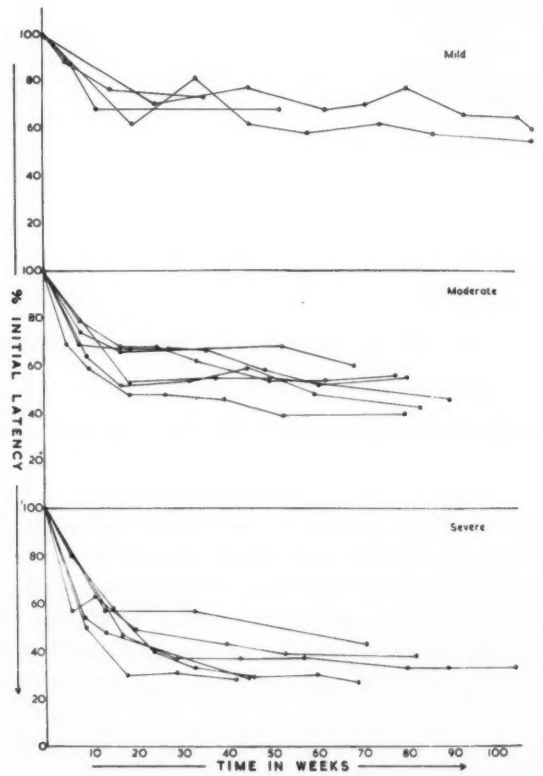

FIG. 6.—Recovery curves from Figs. 3, 4 and 5, replotted with post-operative latency shown as percentage of initial figure in each case.

MOTOR CONDUCTION BEFORE AND AFTER AN INADEQUATE OPERATION

The practical value of latency measurements may be illustrated by the following case history. Owing to the relatively short follow-up period this patient has not been included in the main series.

Case 21.—A male shop assistant aged 43 years was first seen at the Middlesex Hospital in June, 1958. Intermittent paraesthesiae and numbness of median nerve distribution had been present in the right hand for ten years before the onset of increasingly severe pain in the hand caused him to attend hospital. Clinical examination showed no weakness or wasting of the thenar muscles, but there was impairment of sensation to light touch and pin-prick over the median nerve territory in the right hand. He was referred for electrical testing to confirm the diagnosis of carpal tunnel syndrome before surgery was carried out. Pre-operative measurement of motor latency showed a moderately severe delay, the latency being 8·0 msec. (Fig. 7). Operation was carried out on October 21, 1958, by another surgeon using the "blind" method. After operation there was no improvement in his symptoms, and a second conduction test was carried out on March 14, 1959, which showed that no change in motor latency had occurred during the post-operative period. Re-exploration was therefore carried out by Mr. Logue on April 23, a third estimate of nerve conduction immediately before operation showing that there had been no further change in latency. At the second operation the lower part of the flexor retinaculum was found to be intact and was divided under direct vision. Satisfactory relief of symptoms occurred, and it can be seen from Fig. 7 that rapid improvement in motor latency followed this procedure.

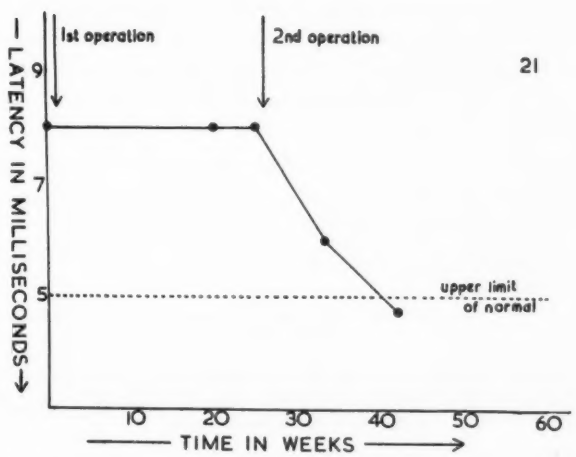

FIG. 7.—Case 21. Motor latency unchanged by first operation, but falls to normal after second operation.

SPLINTING OF THE WRIST

Between December, 1957, and June, 1959, 25 patients were treated by splinting the wrist in the neutral position. Of the 25 patients, 23 wore plaster-of-

Paris volar slabs at night; the remaining two patients wore polythene cylinder splints during the day as well as the night.

The clinical results of splinting were variable. Two patients with severe median nerve damage who were opposed to surgery and therefore given a trial of splinting showed no symptomatic improvement. In the groups with moderately severe or mild nerve damage, three patients were not improved, 14 patients showed partial symptomatic improvement, and three patients underwent complete remission. Of the three patients with normal motor latencies who were splinted, one became worse, one improved partially, and one showed complete remission. Clinical details of individual cases are given by Goodman (1960).

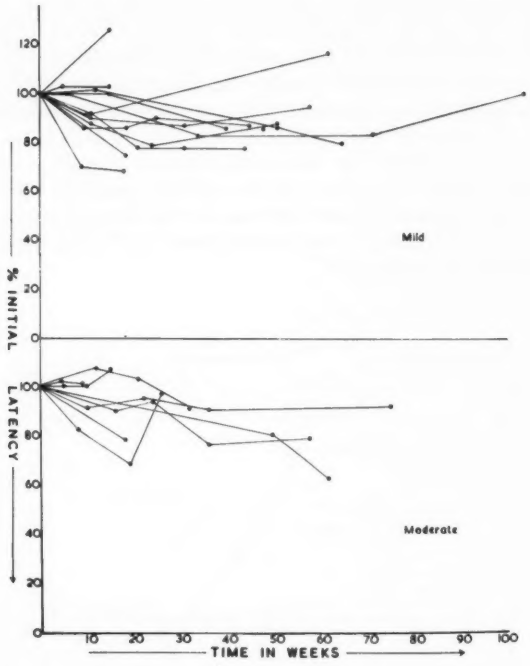

FIG. 8.—Splinting of wrist. Effect of treatment on latency in 20 patients with mild and moderately severe conduction delays. In each case latency during treatment shown as percentage of initial (pre-treatment) figure.

EFFECTS OF SPLINTING ON MEDIAN NERVE CONDUCTION

Group I. Severe Cases.—Two patients with severe nerve damage were treated by splinting. One of these patients was followed up for 11 weeks and the other for 18 weeks, a total of five examinations being performed. In both patients the median nerve was inexcitable at the beginning of treatment and remained so through-

out the follow-up period. For practical reasons it was not possible to continue splinting for a longer period in either patient, and as a result of this experience no further patients with severe delays were treated by splinting.

Group II. Moderately Severe Cases.—In eight cases with moderately severe nerve damage latency measurements were recorded before and during splinting. In all, 30 examinations were carried out for the eight cases, the longest period of follow-up being 75 weeks and the shortest 9 weeks. Results for the whole group are shown graphically in Fig. 8, in which the initial latency for each case has been plotted as 100 and subsequent latencies as a percentage of the initial value. It is clear from Fig. 8 that the results of splinting were not uniform, some of the cases showing improvement in latency and others deteriorating.

Group III. Mild Cases.—Twelve patients with a mild degree of median nerve damage were splinted for periods varying from 15 to 104 weeks. In all, 44 examinations were carried out on the 12 patients. Percentage changes in latency during the period of splinting are shown in Fig. 8. As in the moderately severe group, the results show little uniformity in the response to treatment. Three of the 12 cases showed improvement, while another three deteriorated. The remaining six patients showed little change.

Group IV. Normal Motor Latencies.—Three cases treated by splinting had initial motor latencies within the normal range. These patients were followed up for periods varying between 10 and 21 weeks, a total of seven examinations being carried out. Latency measurements for individual cases are given in Table V, from which it can be seen that in one case latency decreased slightly during the follow-up period, in another there was little change, while in the third case latency increased to a final figure which was outside the normal range.

TABLE V

LATENCY MEASUREMENTS—SPLINTING SERIES: NORMAL MOTOR LATENCIES

Case No.		Pre-treatment	During Treatment	
43	Latency (msec.)	4·8	4·3	
	Time interval (weeks) ..	1	11	
44	Latency (msec.)	4·3	4·1	4·7
	Time interval (weeks) ..	1	12	21
45	Latency (msec.)	4·3	5·5	
	Time interval (weeks) ..	1	10	

SPLINTING FOLLOWED BY OPERATION

There were four patients in the splinted series in whom operative decompression of the median nerve was subsequently carried out. In three of these the post-operative follow-up period was relatively short, and for this reason they have not

been included in the main surgical series. The fourth patient (Case 11) was followed for more than 18 months after surgery, and the full post-operative recovery curve has already been shown in Fig. 4.

In two patients (Cases 11 and 29) symptoms persisted and motor latency actually increased during the period of splinting (Fig. 9). In Case 24 nocturnal pain

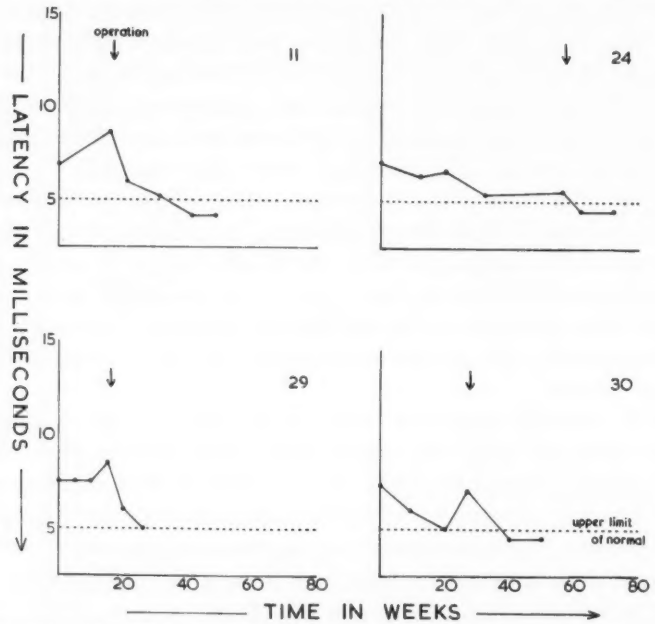

FIG. 9.—Splinting of wrist followed by operation (4 cases). Splinting started immediately after initial latency recording and continued until operation (shown by arrow in each case).

was improved by splinting, but the patient was left with troublesome diurnal paraesthesiae which required operative treatment; the electrical results during the splinting period also showed some improvement, but it can be seen from Fig. 9 that motor latency remained outside the normal range. In Case 30 splinting initially produced relief of both pain and paraesthesiae accompanied by a reduction in motor latency; subsequently the patient's symptoms recurred and latency increased again.

Surgical division of the flexor retinaculum was followed by prompt clinical and electrical recovery in all four patients.

II. Sensory Conduction

In the control subjects investigated by Gilliatt and Sears (1958) stimulation of digital nerves through ring electrodes placed round the index finger produced in every case an afferent volley which could be recorded through surface electrodes

over the median nerve trunk just above the wrist. In their 28 control subjects the amplitude of the recorded potentials varied from 9 to 45 microvolts, and latency to the peak of the main deflection of the action potential varied from 2·5 to 4 msec. for conduction distances of 11 to 15 cm. Gilliatt and Sears found that with increasing stimulus intensity the shock became excessively painful before it was supramaximal for sensory fibres, but it was possible in all cases to choose a stimulus intensity adequate to excite most of the large myelinated fibres, which could still be tolerated by the patient. In some patients with the carpal tunnel syndrome Gilliatt and Sears found that they were unable to record an afferent volley even when the intensity of the stimulus to the finger was raised to the maximum tolerated. In other patients the recorded potentials were of small amplitude and long latency.

In general these findings have been confirmed by the results of the present investigation; sensory action potentials were examined in 33 patients in the present series and were absent in 13. Potentials with an amplitude of less than 9 microvolts or a latency to peak of more than 4 msec. were noted in a further 16. Illustrative records are shown in Fig. 10.

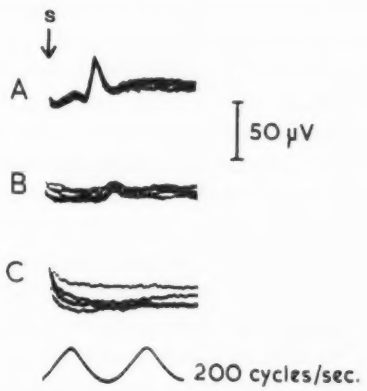

FIG. 10.—Records from median nerve at wrist to show sensory action potentials. Stimulus (S) to index finger at commencement of trace. Records show normal action potential (A), small delayed potential (B), and complete loss of action potential (C). In each record five traces superimposed.

In most patients with severely delayed motor conduction the afferent volleys from the index finger were also absent, but there were some exceptions to this rule. Case 15, for example, showed a long motor delay (9·3 msec.) with an increased sensory latency (7·0 msec.). This may be contrasted with Case 4, in which a long motor latency (15·0 msec.) was associated with a normal sensory action potential. This confirms the well-accepted clinical observation that compression in the carpal tunnel does not always exert its effect equally on the motor and sensory branches of the median nerve.

The examination of sensory nerve action potentials proved to be a rather time-consuming procedure, and for this reason it was not carried out regularly in patients who had been found already to have delayed motor conduction. However, a few patients in the surgical series were studied repeatedly in order to decide whether the final result of operation would be a complete return to normal. Six patients were investigated in this way; in four patients sensory potentials with normal latency and amplitude were found at the end of the follow-up period (Cases 2, 5, 11, and 12R), but in two others (Cases 6 and 7) results were still abnormal at the end of two years.

Sensory conduction was studied in all patients whose motor conduction times were initially normal. There were three patients in the surgical group whose pre-operative figures for motor latency were within the normal range (see Table IV), and in these patients sensory nerve action potentials were examined pre-operatively and subsequently at intervals for periods varying from 60 to 93 weeks post-operatively. In Case 18 latency and amplitude were initially abnormal, but had returned to normal by the end of the follow-up period. In Case 19 latency and amplitude of the sensory potential were initially just within the normal range, but showed an improvement post-operatively. In Case 20 latency and amplitude were both initially within the normal range and showed no change subsequently.

In the splinting series there were again three patients in whom normal values for motor latency were obtained initially (see Table V). Sensory nerve action potentials were examined in these patients before treatment and at intervals during the period of splinting, which varied from 10 to 21 weeks. In Case 43 the sensory action potential was initially increased in latency although normal in amplitude. Latency returned to the normal range during the period of follow-up. In Case 44 the sensory action potential was initially abnormal in both latency and amplitude. Values returned to within the normal range during the period of treatment, but subsequently increased again. In Case 45 the sensory action potential was initially normal in latency but subnormal in amplitude; latency became abnormal during the "splinted" period. It is interesting that this occurred in spite of a satisfactory relief of symptoms.

DISCUSSION

As the object of the investigation was to assess the effects of treatment, there was some bias in the selection of cases towards the inclusion of severely or moderately severely affected patients. In the present series there were among 44 patients only six in whom motor latency was within the normal range; this may be contrasted with the 95 unselected cases examined by Thomas (1960), of which 29 were found to have motor latencies within the normal range. In our six patients with "normal" latencies there were three who showed a reduction in conduction time after treatment, suggesting that a minimal abnormality was in fact present but obscured by the range of variation to be expected in healthy subjects. There is no

doubt that in measuring to the earliest deflection of the evoked muscle action potential one is examining conduction in the fastest, and therefore the least affected, fibres of the nerve trunk. This may be expected to reduce the sensitivity of the test, and it probably explains some of the "normal" motor findings described above in Thomas's series and in our own. In such cases an obviously delayed muscle response is sometimes seen from a few motor units activated by a near-threshold stimulus to the nerve; with increasing stimulus strength this is later obscured by the appearance of units of normal latency. In the present study no account has been taken of delayed responses seen with near-threshold stimuli and abolished by increasing stimulus strength. This phenomenon does, however, offer interesting possibilities for further investigation.

Among the six patients with normal motor latencies before treatment there were three who showed abnormalities of sensory conduction, and in two of them these abnormalities disappeared as the result of treatment. In one further patient sensory conduction was initially normal, but became abnormal during the course of splinting. These figures illustrate the clinical usefulness of recording sensory nerve action potentials which may in mild cases provide the only indication of nerve damage.

The possible causes of prolonged motor latency in this syndrome have been discussed by Thomas (1960). Simpson (1956) believed that the block might have an ischaemic origin, but recent work by Fullerton (1961) indicates that increased susceptibility to ischaemia in this condition bears no relationship to prolongation of motor latency. It is unlikely that the slowing could be accounted for by conduction in regenerating fibres, since an increased latency may remain constant in successive examinations over long periods of time in the absence of treatment (e.g. Case 21). On the other hand, Weiss and Hiscoe (1948) showed in animal experiments that after constriction of a peripheral nerve some nerve fibres could be found distal to the constriction which had survived without degeneration but with considerable reduction in calibre. This finding in experimental animals offers at least a possible explanation of the changes in latency recorded in the present study. Histological evidence of changes in the median nerve in the carpal tunnel syndrome is lacking, except in the case described by Marie and Foix (1913), who found at post-mortem that in the neuroma proximal to the ligament there was a decrease in the number and size of the myelinated fibres, whereas complete degeneration was present distally.

In our surgical series it is extremely gratifying that operation had such a satisfactory result in relieving symptoms and reversing disordered nerve function. So far as motor conduction is concerned, every patient came back into the normal range, although after two years most of the cases in the severe group still showed latencies slightly above the normal mean. It is doubtful whether a more prolonged period of follow-up would have detected alterations of these and one must conclude that complete restoration of normal nerve conduction should not necessarily be

expected in the most severe cases. In this respect it is interesting that in a few patients, who had initially shown gross thenar wasting, muscle bulk failed to recover completely during the period of observation. However, these points do not detract from the general conclusion that an adequate operation is such a highly satisfactory procedure in the carpal tunnel syndrome that for all practical purposes it constitutes a curative treatment for this condition.

Although the results of splinting were variable and often disappointing compared with the uniformly good results of surgery, several points of interest have emerged. In the first place, our results confirm the observations of Heathfield (1957) that some patients with the carpal tunnel syndrome do obtain symptomatic relief with this form of treatment. This occurred in a much higher proportion of Heathfield's cases than our own, a difference which is almost certainly explained by the fact that Heathfield's series contained only mildly affected patients, those with thenar wasting or persistent sensory loss being rejected. It is remarkable that splinting the wrist joint at night with free movement during the day should be sufficient to produce symptomatic relief, and it might be suggested that the beneficial effect is due not only to immobilization of the wrist but also to the fact that the patient is prevented from sleeping on the affected side in bed.

In general, the use of conduction tests in the present series of patients has proved highly satisfactory from the practical point of view. It has provided detailed information about the state of the median nerve during and after treatment, and, while the value of accurate history-taking and careful clinical examination should not be underestimated, there is no doubt that the physicians and surgeons concerned with the patients in this series have tended to place increasing reliance on the results of electrical tests.

The investigation is seen to particular advantage in the assessment of the difficult patient when operation has been carried out without clinical benefit or when there is a recurrence of symptoms. This situation arose in several cases, three patients in our surgical series having previously had operations which were registered as technically satisfactory by the surgeons concerned, although symptoms continued. Motor latency measurements in all three cases showed persistence of electrical abnormalities. There were four patients in our splinting series who finally required surgery, and in these cases also the decision to operate was made easier by the repeated examination of nerve conduction.

SUMMARY

Median nerve conduction was studied in a series of 44 patients; in three patients both hands were examined. Motor conduction between the wrist and thenar muscles was normal in six cases. In the remaining 41 cases conduction was slowed, resulting in a delayed muscle response to nerve stimulation.

Surgical division of the anterior carpal ligament was carried out in 23 cases. Serial examinations of motor conduction for up to two years after operation showed

satisfactory recovery of conduction in all cases, the recovery time depending on the severity of the initial lesion.

Splinting the wrist was tried in 25 cases and motor conduction was examined repeatedly during treatment for periods up to two years. Results were variable, and in some patients abnormalities of conduction were seen to increase during the course of treatment.

The examination of conduction in the sensory fibres of the median nerve was found to be particularly useful in mildly affected patients in whom motor studies gave normal results.

Conduction tests proved to be of considerable practical value in the management of the patients studied.

Acknowledgments

We are particularly grateful to Mr. Valentine Logue for his enthusiastic collaboration and for allowing us to quote operative findings from his cases.

A personal grant to H. V. G. from the Sir Halley Stewart Trust is gratefully acknowledged. We are also indebted to the Medical Research Council for a grant towards the cost of apparatus, and to the Clinical Research Committee of the Middlesex Hospital for providing facilities.

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ORIGINAL PAPER

VISCERO-SOMATIC REFLEXES*

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Before discussing some aspects of the anatomy of viscero-somatic reflexes it is necessary to refer briefly to the nomenclature of the autonomic nervous system. Following Gaskell and Langley, many people refer to the autonomic nervous system as a purely efferent system. In part, this view is held for reasons of terminological convenience, but it also reflects the concept of the autonomic nervous system as a separate part of the central nervous system. Since Gaskell and Langley's time, however, it has become increasingly clear that the autonomic nervous system is intimately linked with the rest of the central nervous system. One has only to think of reflexes such as the cough reflex, which is mediated on the afferent side by fibres in the vagus nerve and on the efferent side by fibres in the nerves to the respiratory and laryngeal muscles, to see how closely the autonomic and somatic nervous systems are linked. Respiration also involves movement accomplished by striated muscle innervated from the somatic nervous system, together with changes in bronchiole size and pulmonary blood-flow which are effected through the autonomic nervous system. Similarly, activation of muscle proprioceptors produces hyperpnoea and an increase in heart rate and arterial blood-pressure through autonomic efferents (Harrison, Calhoun, and Harrison, 1932; Comroe and Schmidt, 1943), and the responses of the pupil and peripheral blood-vessels to painful stimulation of the skin are well known. The integration of the autonomic with the somatic nervous system occurs at all levels in the central nervous system—that is, at the level of peripheral nerves, the spinal cord, the brain-stem, and subcortical and cortical centres. For this and other reasons there is thus little to be gained, and quite a lot to be lost, by excluding afferent nerve-fibres from the viscera, blood-vessels, etc., in the definition of the autonomic nervous system. In fact, it seems likely that the division of the nervous system into autonomic and somatic components will eventually disappear altogether.

So far as we know at the moment the cells of origin of autonomic afferent nerve-fibres included in the sympathetic nervous system are found in posterior spinal-root ganglia, and those included in the parasympathetic nervous system have their cells of origin in the root ganglia of the third, fifth, seventh, ninth, and tenth cranial nerves, groups of cells near the nuclei ambigui, and in the dorso-lateral part of the mid-sacral ventral columns.

The general anatomical characteristics of autonomic afferent nerve-fibres are as follows. Some of them go by short pathways to the spinal cord, and others go by much longer pathways to the brain-stem. Thus pelvic afferent nerve-fibres from the

* Paper read at the Annual Meeting of the British Association of Physical Medicine, London, April, 1961.

bladder go directly to the sacral segments of the cord via the dorsal spinal roots, whereas afferents from the colon ascend in the vagus nerves to the dorsal vagal nuclei. In the spinal cord some autonomic afferents are linked in the region of their segments of entry with cells in the intermedio-lateral columns of grey matter and constitute the pathway for local spinal reflexes. The spinal segments concerned with these local reflexes in human beings are, according to Ingram (1960):

Head and neck	T1–5
Upper extremity	T2–9
Lacrimal glands (sympathetic)	T1–3
Vasomotor responses, piloerection, and sweating:	
Upper trunk	T4–9
Lower trunk	T9–L2
Lower extremities	T12–L2
Pupillo-dilatation	C8–T1
Cardiac acceleration	T2–T6
Abdominal viscera	T4–L2
Genito-urinary and recto-anal (sympathetic) responses	L1–L2

Other autonomic afferent nerve-fibres spread up and down in the cord for several segments and their central fibres ascend in the white matter of the spinal cord to centres in the brain-stem. The ascending pathways of these fibres in the spinal cord are not yet clearly defined. One of the most precise investigations was that of Downman and Evans (1957), who examined the ascending splanchnic pathways in the cat (Fig. 1). These authors concluded that their results "support the view

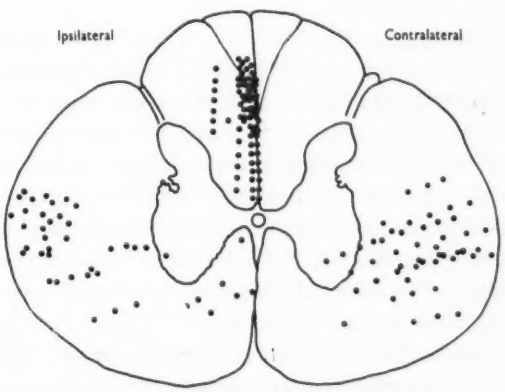

FIG. 1.—Ascending pathways of splanchnic nerves in C2 and 3 levels of spinal cord of cat. (Downman and Evans, 1957.)

that visceral afferent fibres ascend the spinal cord in the same regions as homologous somatic afferents". However, there is evidence from the results of cordotomy that autonomic afferent fibres also ascend in the ventral white matter.

Autonomic afferent fibres have been traced to the reticular formation in the brain-stem, to the hypothalamus, to the amygdala, and to the thalamus.

It is known that, in animals, the vagus and splanchnic nerves project from these subcortical areas to the cerebral and cerebellar cortices. The cerebral cortical areas in the cat are shown in Fig. 2. It is interesting to note the close proximity of these cortical projections of autonomic afferent fibres to the somatic motor areas.

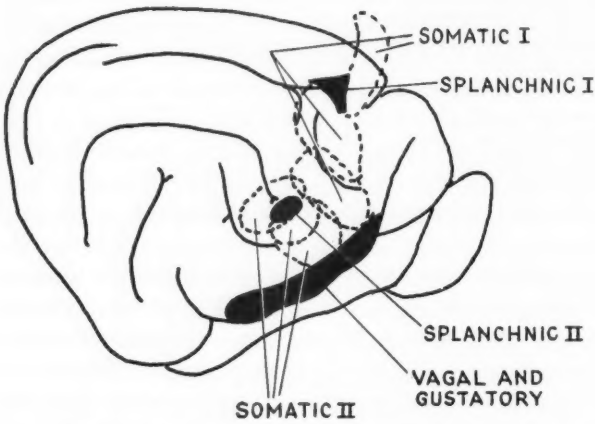

FIG. 2.—Afferent cortical areas in cat (modified from Dell, 1952).

The general physiological characteristics of autonomic afferent fibres are as follows. We have no information, as yet, of their being separable into modalities with discrete spinal pathways such as pain, touch, etc. It may be that no such subdivision of autonomic afferent fibres is possible, but it may be that we have not studied this aspect of the autonomic nervous system sufficiently. However, a subdivision of the functions of autonomic afferent fibres is possible to some extent. There are autonomic afferent fibres (*a*) concerned with the control of the circulation, respiration, and glandular activity—e.g. the carotid sinus, Hering–Breuer, and Bainbridge reflexes; (*b*) concerned with normal visceral sensation—e.g. hunger, distension of the gastro-intestinal tract, sex, etc.; (*c*) concerned with visceral pain; and (*d*) concerned with adjustments of muscle tone and reflex excitability in response to (*a*), (*b*), and (*c*) and in response to changes in posture. I will deal with the last two aspects of autonomic function, which mainly concern the effects of autonomic *afferent* fibres on somatic reflexes, in some detail as the other functions are relatively well known.

Before discussing these, however, I would like briefly to mention some of the effects of *efferent* autonomic activity on somatic function. The so-called "Orbeli effect" (Orbeli, 1925) is well known. When a nerve–muscle preparation is stimulated

to the point of fatigue subsequent stimulation of the sympathetic nerve supply to the muscle restores the response of the muscle. This effect of sympathetic nerve stimulation is not due to changes in the blood-supply to the fatigued muscle, as the effect is present in the exsanguinated frog (Maibach, 1928; Labhart, 1929). It is probably due to an indirect effect of liberated adrenaline on the muscle. Apart from such possible local effects as that described by Orbeli, it seems unlikely that

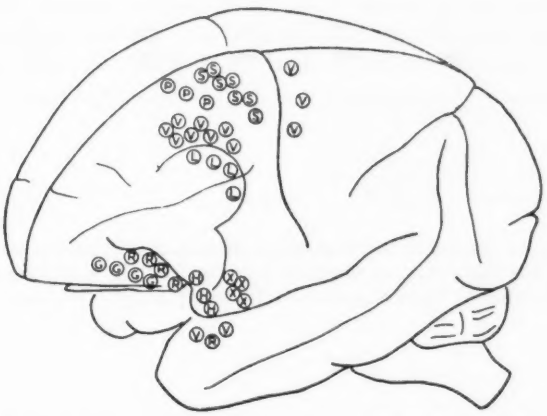

FIG. 3.—Cortical areas in monkey from which autonomic effects have been elicited by chemical or electrical stimulation. G=gastro-intestinal; H=cardiac; L=lacrimation; P=pilomotor; R=respiratory; S=sudomotor; X=salivary; V=vasomotor effects.

the proximity of certain cortical areas producing autonomic effects to somatic motor areas has evolved by chance. Fig. 3 shows the areas in the monkey cortex from which autonomic effects have been elicited by electrical or chemical stimulation. Figs. 4, 5, and 6 show the effects of stimulating various cortical areas on blood-pressure and respiration and gastric secretion.

How these cortical autonomic areas may influence somatic function is illustrated by experiments I performed on human volunteers. Contracting the muscles of the forearm while keeping the joints of the arm fixed is accompanied by vasodilatation in the muscles which can be demonstrated plethysmographically. I was able to show that the mere act of thinking about contracting the forearm muscles without actually contracting them is also accompanied by vasodilatation. This vasodilatation can be produced only by cortical activity. It is reasonable to assume that cortical activity not only influences, for example, somatic musculature, but also modifies peripheral autonomic activity, which in its turn influences centrally and peripherally controlled somatic function. It is interesting to note that Landau (1953) was able to modify the discharge rate of the vagus and splanchnic nerves by electrical stimulation of the pyramidal tracts in cats (Fig. 7).

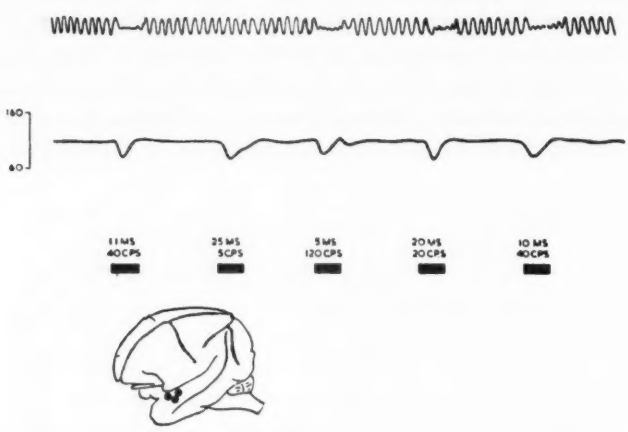

FIG. 4.—Effects on respiration (top tracing) and arterial blood-pressure (bottom tracing in mm. Hg) of monkey on stimulation of temporal lobe (see inset). Signal marks show duration of stimulating pulses (5–25 msec.) and their frequency (c.p.s.). (Wall and Davis, 1951.)

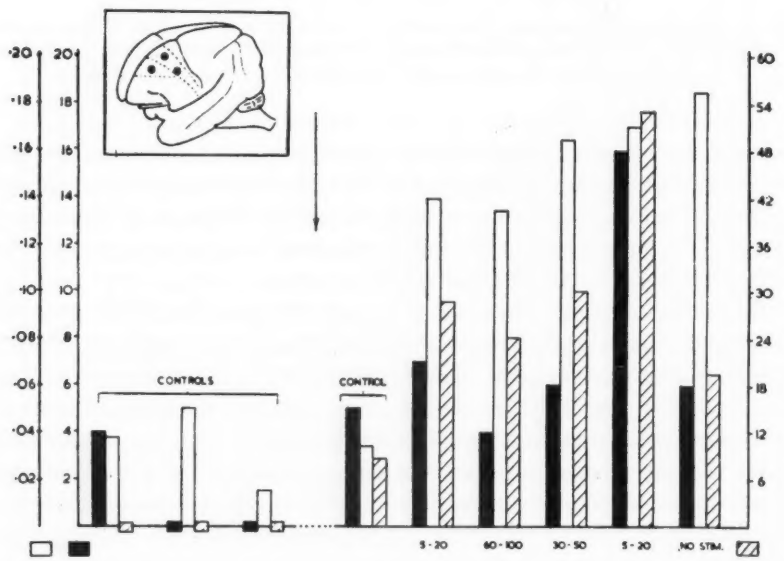

FIG. 5.—Effects of stimulation of cortical areas 6 and 8 (see inset) on volume (black columns) and pepsin (open columns) and HCl (hatched columns) content of gastric juice of dog. Arrow marks point when stomach was aspirated and pylorus tied off, and figures below columns show frequency of stimulation in c.p.s.

Some effects which may be due to the influence of autonomic nervous activity on the general function of the somatic nervous system are matters of everyday observation. We have all seen the hyperkinesis of the child with a full bladder, and lethargy after eating is frequently seen in adults and tends to become more marked with increasing age. It is interesting that in children one often sees the reverse effects of eating, and this can be a source of familial strife. The small child is often very lethargic when hungry and becomes very active after eating. There is some evidence that these effects of hunger and eating are not solely due to the blood-sugar level, but are also due to the rate of autonomic afferent discharge from the stomach as a result of its state of distension. Thus, M. H. Evans and I showed that distension of the stomach facilitates reflex activity in the L6 segments of the spinal cord of the chloralosed cat.

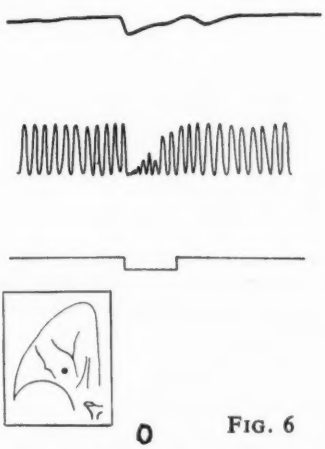

FIG. 6

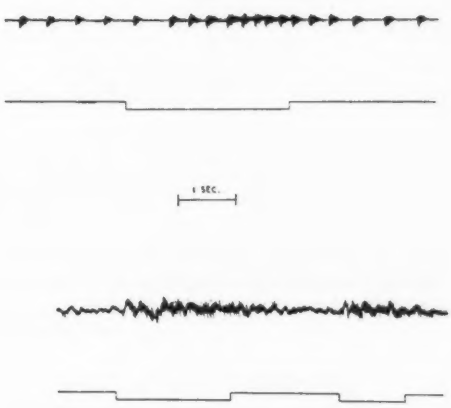

FIG. 7

FIG. 6.—Effects on arterial blood-pressure (top tracing showing an initial decrease of 60 mm. Hg) and respiration (bottom tracing) of monkey of electrical stimulation of posterior orbital gyrus (see inset).

FIG. 7.—Effects on discharge recorded from vagus (upper tracing) and abdominal sympathetic nerves (lower tracing) of stimulation of pyramidal tracts in medulla of cat. (Landau, 1953.)

Much of the work which has been done on the effects of stimulation of abdominal viscera on somatic motor function has been aimed at elucidating the mechanisms involved in visceral pain. Thus Mackenzie regarded the abdominal rigidity associated with visceral disease as a reflex phenomenon, and he obtained Sherrington's help to study it. Sherrington produced movements of the abdominal wall in cats by stimulating the bile-duct or the central end of the superior mesenteric nerve. Later, Miller and Simpson (1925) and Miller and Waud (1925) described contractions of the abdominal and hind-limb muscles in cats as a result of

mechanical stimulation of abdominal viscera and electrical stimulation of mesenteric nerves. In 1937 Dusser de Barenne and Ward described flexion of the hind-limbs of cats and a powerful inhibition of the knee-jerk as a result of distension of the gall-bladder, the urinary bladder, and the ileum.

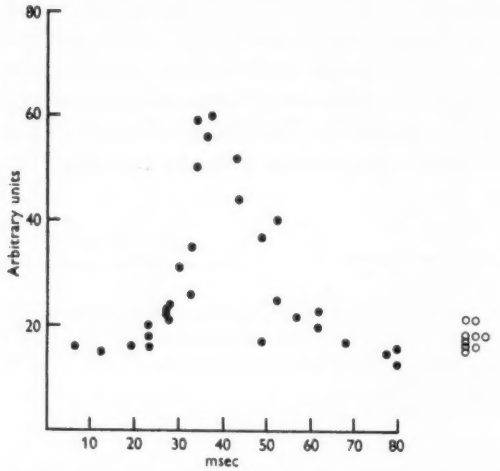

FIG. 8.—Graph of amplitude (in arbitrary units) of monosynaptic responses recorded from L6 ventral root evoked by stimulation of L6 dorsal root, plotted against interval between dorsal root and sympathetic chain stimuli. O=test responses; ●=responses to interaction between dorsal root and sympathetic stimulation.

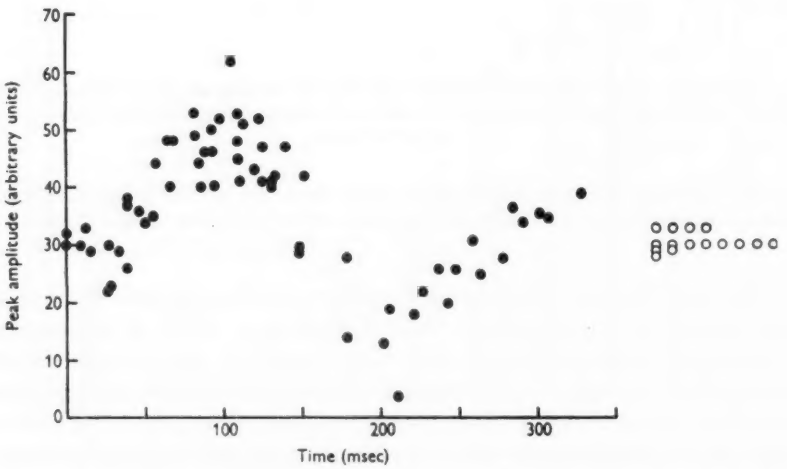

FIG. 9.—As in Fig. 8, but showing results of interaction of dorsal-root and splanchnic-nerve stimuli.

More detailed electrophysiological studies of the effects of stimulating abdominal viscera on somatic reflexes have been done by Downman and McSwiney, Downman, Eble, and Evans, and me. Downman (1951, 1952, 1953, and 1955) studied the effects of electrical stimulation of the splanchnic nerve on somatic reflexes recorded from intercostal and lumbar nerves in spinal and decerebrate cats. It is difficult in summary form to do justice to Downman's careful work, but some of the more interesting results he found include the fact that the reflex discharge produced by splanchnic stimulation in spinal cats is of long duration and irradiates over wide areas of the spinal cord. The routes of this splanchnic irradiation are a fast extraspinal path in the sympathetic chain of the same side and a slower intraspinal route to both sides. Downman's results support Amassian's (1951) view that the effects on somatic motoneurones of stimulation of visceral afferents are mediated by the A gamma-delta group of fibres in the visceral nerves.

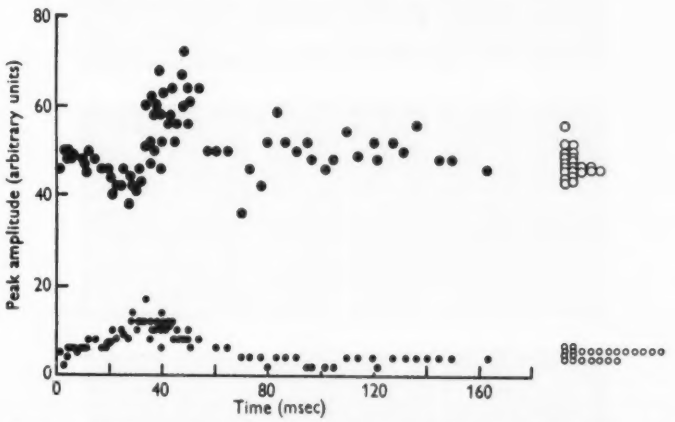

FIG. 10.—As in Figs. 8 and 9, but showing results of interaction of dorsal-root and pelvic-nerve stimuli on monosynaptic (upper) and polysynaptic (lower) reflexes.

M. H. Evans and I (1958, 1960) examined the effects of electrical stimulation of the vagus, splanchnic, pelvic, and hypogastric nerves and the thoracic sympathetic chain on monosynaptic and polysynaptic reflexes elicited from the lumbar segments of the spinal cord of anaesthetized, spinal and decerebrate cats. Monosynaptic reflexes were produced by single-shock stimulation of either somatic afferents from muscles or dorsal spinal roots and were recorded either from somatic muscle efferents or from the ventral spinal roots. The analysis of these results proved rather difficult, as both facilitation and inhibition of monosynaptic reflex arcs was produced by stimulation of visceral afferents.

Fig. 8 shows a graph of the amplitude, in arbitrary units, of monosynaptic reflexes recorded from the L6 ventral root and evoked by stimulation of the L6 dorsal root in the anaesthetized cat when the central cut end of the sympathetic

chain in the thorax is also stimulated. The abscissa shows the interval between the preceding sympathetic stimulus and the stimulus producing the monosynaptic response. When the stimulus to the sympathetic chain precedes that to the dorsal spinal root by about 25 to 50 msec. the monosynaptic response is increased up to

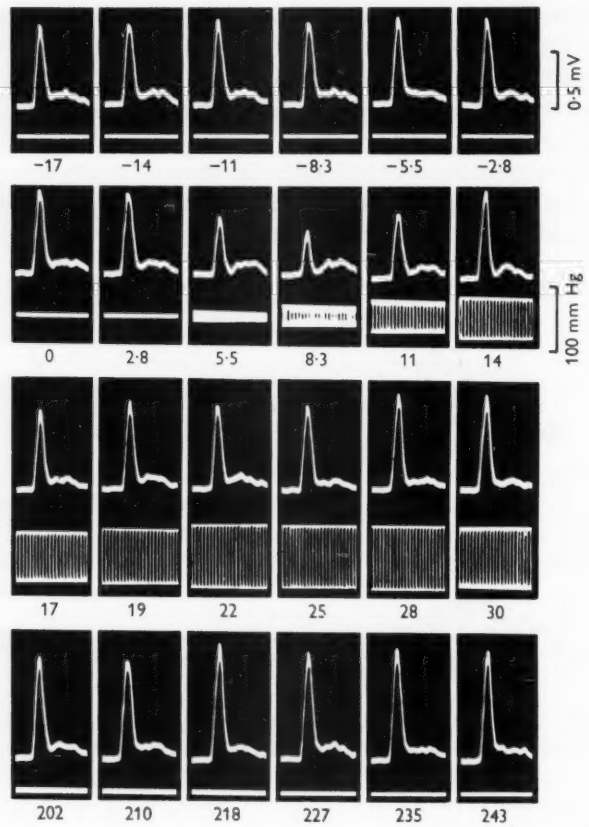

FIG. 11.—Monosynaptic with small early polysynaptic reponses recorded from 7th lumbar ventral root after stimulation of ipsilateral 7th lumbar dorsal root in a precollicular decerebrate cat. Lower beam records intravesical pressure. Top row, control responses with bladder empty; second and third rows, distension of bladder with 50 ml. fluid; bottom row, after emptying bladder. Figures indicate time in seconds before and after start (0) of distension.

400% of its original amplitude. Stimulation of some visceral afferents will also produce inhibition of monosynaptic responses. Fig. 9 hows the effects of preceding the dorsal-root stimulus by a conditioning stimulus to the left splanchnic nerve in the decerebrate cat. At intervals between the two stimuli of approximately 50 to 150 msec. the monosynaptic response was facilitated and at intervals of 150 to

250 msec. it was inhibited. Similar effects of stimulation of the pelvic nerve on both monosynaptic and polysynaptic reflexes are shown in Fig. 10. Evans and I concluded that (*a*) single shock stimuli to visceral afferent nerve fibres both facilitated and inhibited monosynaptic reflexes; (*b*) such stimuli facilitated short-latency polysynaptic reflexes, but inhibited long-latency polysynaptic reflexes; and (*c*) these excitability changes were commonly seen on stimulation of afferent fibres in the splanchnic and pelvic nerves and in the sympathetic chain, but were seldom seen on stimulation of the vagus or hypogastric nerve.

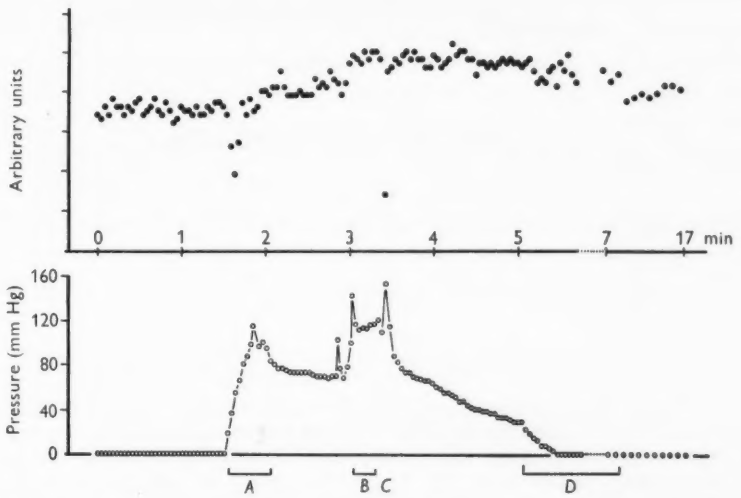

FIG. 12.—Same experiment as Fig. 11. Height of monosynaptic reflexes (including those illustrated in Fig. 11) plotted on upper graph and intravesical pressures on lower graph. *A*, 50 ml. fluid into bladder; *B*, additional 20 ml. into bladder; *C*, spontaneous bladder contraction; *D*, withdrawal of 70 ml. from bladder. Note compressed time scale for last 11 points.

In an attempt to see the functional significance of these viscerosomatic reflexes Evans and I (1958, 1959) examined the effects of distension of the viscera on mono- and poly-synaptic reflexes. The results of some of these experiments are illustrated in Figs. 11 and 12. Fig. 11 shows monosynaptic and short-latency polysynaptic reflexes recorded from the L7 ventral root after stimulation of the L7 dorsal root in a decerebrate cat. The lower beam records the pressure in the bladder, which was distended with Locke solution. It can be seen that the monosynaptic response is first inhibited and then facilitated by distension of the bladder. This is shown graphically in Fig. 12. One of the most interesting features of these experiments was that distension of the bladder produced facilitation of monosynaptic reflexes which lasted up to 20 minutes after the intravesical pressure returned to normal.

We studied the effect of distension of the bladder on the tone and reflex contractions of somatic muscles, recording the latter by means of a myograph. Some of these effects are quite dramatic. In the experiment illustrated in Fig. 13 the changes in tone and reflex contractions of a tibialis anterior muscle and its crossed extensor in response to distension and spontaneous contraction of the bladder in a decerebrate cat are shown. The reflex contractions were produced by single-shock stimulation of the medial popliteal nerve on the same side as the tibialis anterior

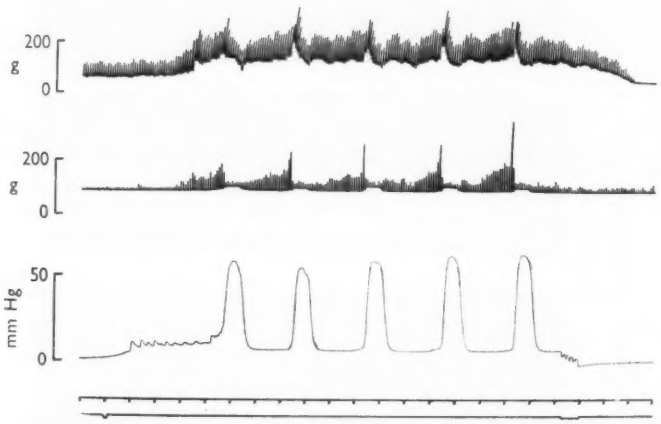

FIG. 13.—Upper tracing: reflex contractions of crossed extensor, recorded from left gastrocnemius–soleus tendon following stimulation of right medial popliteal nerve in a precollicular decerebrate cat. Middle tracing: flexor contractions of right tibialis anterior muscle. Lower tracing: intravesical pressure (mm. Hg). At the first signal mark one channel of the bladder catheter was connected to a reservoir of Locke's solution, which was run in slowly, while the true intravesical pressure was recorded through the other channel of the catheter; at the second signal mark the reservoir was disconnected and 45 ml. fluid was emptied from the bladder. Time marker, 1 min.

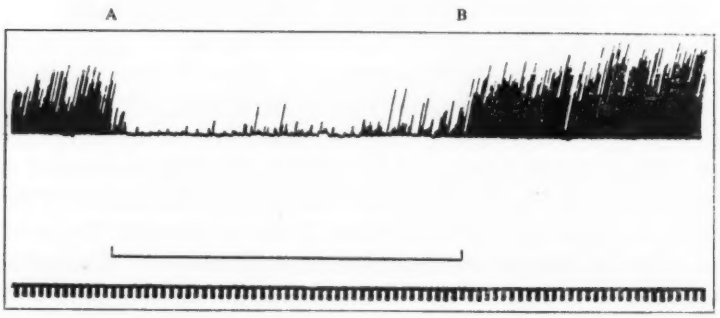

FIG. 14.—Reflex extension of whole leg recorded at ankle, following stimulation of contralateral medial popliteal nerve in a chloralosed cat. At A the bladder was filled with fluid from a reservoir 10 cm. above the bladder; at B 22 ml. of fluid was withdrawn. Time marker, 10 sec.

muscle. The changes in muscle tone and reflex excitability of these hind-limb muscles are most marked and are not due to the changes in respiration and arterial blood-pressure which accompany distension of the bladder. It is also possible to produce inhibition of hind-limb reflexes by bladder distension. In Fig. 14 the reflex extension of the whole leg recorded at the ankle following stimulation of the contralateral medial popliteal nerve is recorded in the anaesthetized cat. At A the bladder was filled and reflex extension was inhibited, and at B the bladder was emptied and the reflex extension was facilitated. A particularly interesting feature of these experiments was that, when the bladder was distended to a pressure which would be expected to produce pain in the conscious animal, hind-limb flexor muscle tone was increased and hind-limb reflexes were inhibited—a mechanism which is probably relevant to the attitude of flexion and abdominal rigidity seen in visceral disease. Evans and I (1959) suggested that the effects of bladder distension on somatic reflexes may be mediated by bladder tension receptors acting on the small motor-nerve (gamma efferent) system, and that viscero-somatic reflexes are subject to modification and control from supraspinal centres.

Abdullah and Eldred (1959) examined the activity in gamma efferents induced by distension of the bladder in spinal cats. Some of their results are shown in Fig. 15, from which it can be seen that the rate of discharge of the gamma efferents

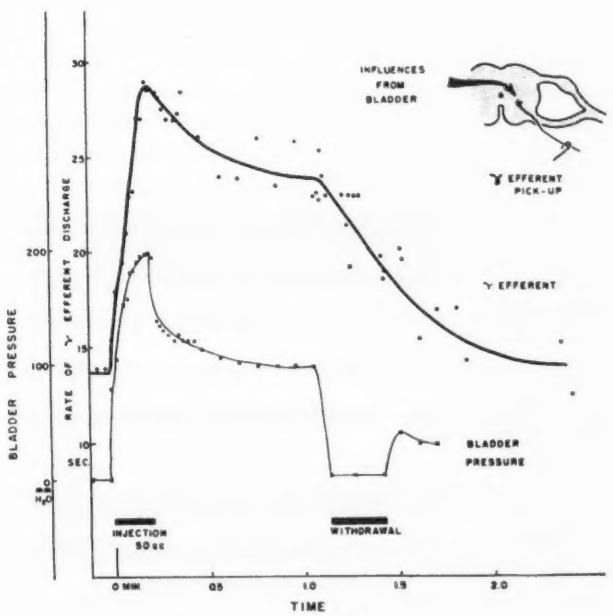

FIG. 15.—Abdullah and Eldred's (1959) results of effects of changes in bladder pressure on small motor nerve discharge.

closely follows the bladder pressure. We thus have evidence that visceral activity can alter the discharge rate from muscle spindles.

Tolle, Feldman, and Clemente (1959) have also shown in cats that visceral afferent impulses recorded in the spinal cord could be inhibited by simultaneous stimulation of the ventromedial reticular formation. The areas in the medulla from which they inhibited visceral afferent responses are shown in Fig. 16.

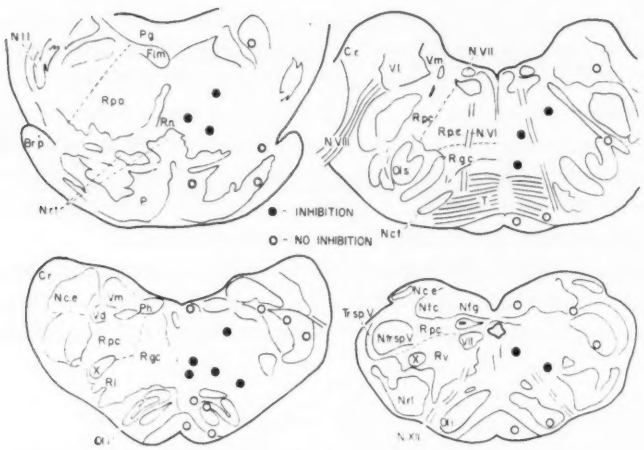

FIG. 16.—Areas in medulla of cat stimulation of which produces inhibition of visceral afferent impulses recorded in spinal cord (Tolle *et al.*, 1959).

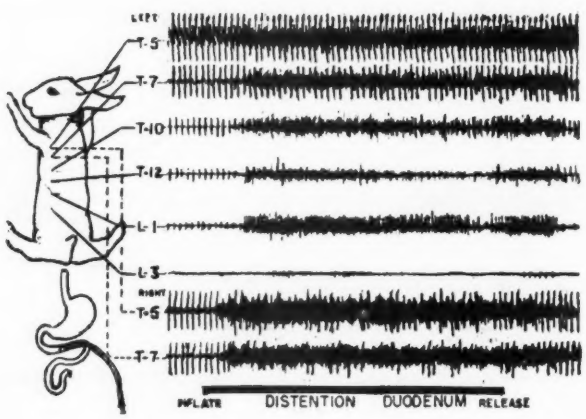

FIG. 17.—Changes in E.M.G. recorded from paravertebral muscles produced by distending the duodenum (Eble, 1960).

Recently Eble (1960) has examined the effects on the electromyogram, recorded from paravertebral muscles of cats, of stimulating the renal pelvis, ureter, small intestine, and Fallopian tube. He found that such visceral stimulation produces widespread and long-lasting reflex contractions of the paravertebral muscles. Fig. 17 shows the bilateral reflex responses of paravertebral muscles to distension of the duodenum.

So far I have discussed the effects of abdominal visceral activity on the somatic nervous system, but there is also evidence of the effects of such structures as the carotid sinus on somatic reflexes. Koch showed that increasing the pressure in the carotid sinus of the conscious dog causes bodily movements to stop, the eyelids to close partially, the head to hang, etc., so that the animal appears to sleep. Dell (1956) has shown more recently that autonomic afferent impulses coming from the carotid sinus affect the animal's state of arousal by acting on the midbrain reticular formation.

An aspect of the interaction between the autonomic and the somatic nervous system which has not so far been mentioned is the effect of somatic stimuli on autonomic function. Vasoconstriction and pupillo-dilatation in response to painful stimuli of somatic structures are well known. Similar examples of somato-visceral reflexes are the decrease in heart-rate produced by pressure on the eyes, apnoea produced by sudden cold stimuli, erection in response to tactile stimulation, etc. An aspect of somatovisceral reflexes which could well repay more study is the effect of somatic stimuli on the abdominal viscera.

Freude and Ruhmann (1926) claimed from X-ray studies that cold stimuli in the epigastrium reduce gastric activity and that hot stimuli to the same region increase it. Ruhmann (1927a, b, c) also suggested that mechanical and chemical stimulation of the abdominal skin produces similar changes, and that in fact cutaneous hyperaemia is accompanied by visceral hyperaemia and cutaneous ischaemia is accompanied by visceral ischaemia. These latter conclusions received support from the photographic and plethysmographic evidence of Kuntz and Haselwood (1940). These visceral responses to cutaneous stimuli are of a segmental character, and there is strong evidence that they are not produced by the direct effects of heat or cold on the viscus.

From all the experiments described it is possible to suggest a schema of autonomic–somatic interaction for which the experimental evidence varies in extent and quality.

SUMMARY

Evidence is presented that: (1) Autonomic afferent impulses both excite and depress somatic reflexes at the spinal level. (2) These autonomic afferent effects at the spinal level are modified by supraspinal control both of the autonomic efferents and the somatic efferents. (3) Autonomic afferents ascend to subcortical and cortical

centres and modify somatic reflexes at these levels. (4) Somatic afferent impulses modify autonomic afferent and efferent activity at all levels of the nervous system. (5) Efferent autonomic activity modifies somatic reflexes both locally and by means of descending autonomic efferents.

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ORIGINAL PAPER

CORTICOSTEROID MYOPATHY*

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THE term "myopathy" has been applied to a variety of conditions involving muscle fibres and causing pain, weakness, wasting, stiffness, or varying combinations of these. The myopathies are divided into the heredo-familial and the acquired, the latter including several endocrine and metabolic conditions. There is considerable evidence to suggest that excess of circulating adrenocortical hormone (cortisol) or one of its synthetic derivatives may cause myopathy; reports relating to this are summarized in the Table on page 172. The predominance of cases involving triamcinolone and dexamethasone has suggested the possibility that the fluorine atom present in these corticosteroids may be responsible. However, it must be remembered that these more powerful, halogenated corticosteroids have been administered to larger numbers of patients, and often in greater equivalent dosage, than prednisolone.

Myopathy may also occur as an integral feature of several musculo-skeletal diseases, including rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, dermatomyositis, polymyositis, and scleroderma. Steinberg and Wynn Parry (1961) found electromyographic evidence of myopathy in 79 out of 93 patients suffering from rheumatoid arthritis, and stated that few of these patients had been treated with corticosteroids.

The purpose of this paper is to present the case histories of five patients who developed myopathy during the course of long-term corticosteroid therapy, and who were seen and investigated in the Department of Physical Medicine during the period February, 1960, to February, 1961. Brief histories of two patients suffering from rheumatoid arthritis who developed myopathy, although they had not received corticosteroid therapy, are also given. The electromyographic pattern of a myopathy was seen in all the patients, and an abnormal tracing is shown in comparison with a normal electromyogram in Plate V, Fig. 1. The principal features indicating a myopathy are an interference pattern that is only slightly reduced; an increased number of small potentials with reduced amplitude and duration; an increased number of short-duration polyphasic potentials, and a "shift to the right" shown by the frequency analyser, representing an increase in the high-frequency components of more than 420 cycles per second.

* Paper read at the Annual Meeting of the British Association of Physical Medicine, London, April, 1961.

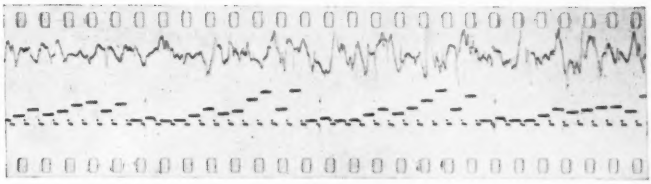
CORTICOSTEROID MYOPATHY: HISTORICAL REVIEW

	Year and Author	Reported Findings
Cushing's syndrome	1932 Cushing	Weakness in 5 out of 12 cases of "basophil adenoma of pituitary"
	1952 Plotz *et al.*	Weakness in 27 out of 33 cases
	1959 Müller and Kugelberg	Myopathy in 6 out of 7 cases
Experimental steroid administration	1945 Kendall, E. C.	Muscle lesions in cortisone-fed rabbits
	1951 Germuth *et al.*	„ „ „ „
	1956 Ellis	„ „ „ „
	1960 Kendall, P. H.	Muscle lesions in dexamethasone-fed rats
Cortisone	1959 Perkoff *et al.*	Weakness in 3 cases of blood disorders
Fludrocortisone (9-α-fluorohydrocortisone)	1959 MacLean and Schurr	Weakness ascribed to fludrocortisone given after removal of craniopharyngioma
Prednisone (Δ-1-cortisone)	1959 Harman	Weakness in 1 case of leukaemia and 1 case of systemic lupus erythematosus
	1959 Perkoff *et al.*	Weakness in 4 cases of blood disorders
Triamcinolone (9-α-fluoro, 16-α-hydroxyprednisolone)	1958 Freyberg *et al.*	Weight loss in 5 out of 89 cases of rheumatoid arthritis
	1958 Dubois	Weakness in 6 cases of systemic lupus erythematosus
	1959 Kendall and Hart	Myopathy in 5 cases of rheumatoid arthritis
	1959 Williams, R. S.	Myopathy in 2 cases of "collagen" disease and 1 case of asthma
Dexamethasone (9-α-fluoro, 16-α-methylprednisolone)	1959 Duvenci *et al.*	Weakness in 2 cases of asthma
	1959 Walton	Weakness in 2 cases of allergic disorders
	1959 Williams, G. T.	Weakness in 1 case of rheumatoid arthritis
	1960 Golding and Begg	Myopathy in 1 case of asthma
	1960 Syme	Weakness in 1 case of temporal arteritis
	1960 Bergman	Weakness in 5 children treated with dexamethasone

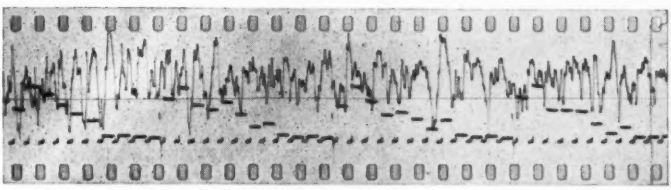
CASE HISTORIES

CASE 1

This was a 60-year-old man suffering from asthma, whose case history has already been reported (Golding and Begg, 1960). Corticosteroid therapy was started in October, 1959, with prednisolone 30 mg. daily for two months, then changing to dexamethasone 2·25 mg. daily for five months. After three months of dexamethasone therapy weakness and wasting of the proximal limb muscles, chiefly the thighs, developed. Voltage–duration



A



B

FIG. 1.—A. Normal electromyogram: unit potentials on volition, with frequency histogram. B. Case 1: motor unit potentials on volition, with frequency histogram.

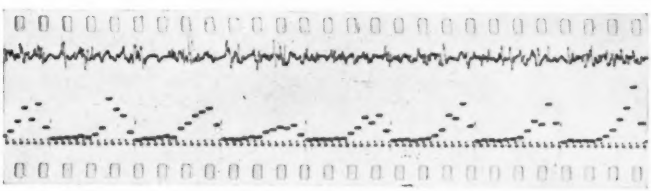


FIG. 2.—Case 2: motor unit potentials on volition, with frequency histogram.

All electromyograms were calibrated at 300 μV.

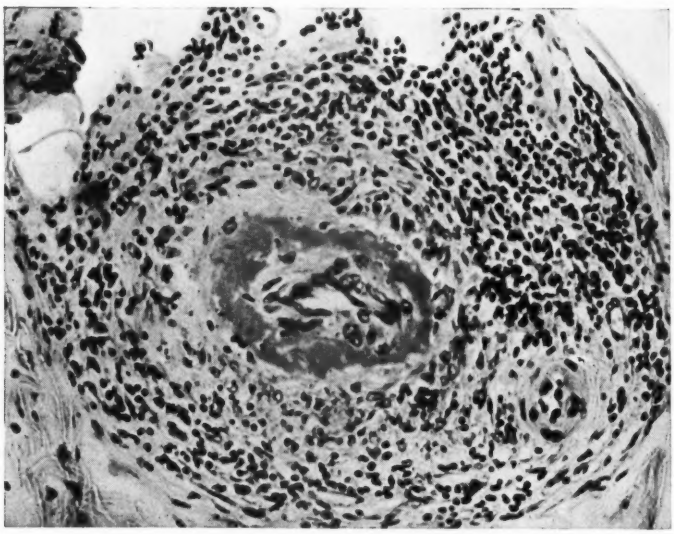


FIG. 3.—Artery from biceps brachii showing inflammatory and necrotic changes. (H. and E. ×190.)

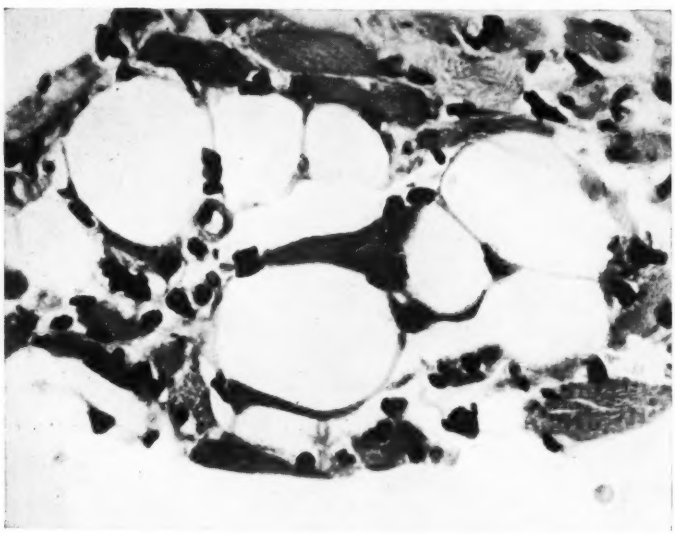


FIG. 4.—Gastrocnemius muscle showing small voluntary muscle fibres distorted by fat cells. (H. and E. ×550.)

curves from both quadriceps were normal, but electromyograms (EMG) showed a myopathic pattern. Urinary creatine excretion was raised to 615 mg. in 24 hours (normally 25 to 100 mg.). Urinary creatinine excretion was 924 mg. in 24 hours, the normal upper limit being 1,000 mg. Serum aldolase was 8 Bruns units (normal, 1 to 9 units) and serum glutamic oxalacetic transaminase (S.G.O.T.) 27 sigma Frankel units. The electrocardiogram (ECG) was normal.

Muscle biopsy showed a small amount of nuclear proliferation and clumping together with a few short chains of hyperchromatic nuclei. There was no fibre necrosis. Examination of muscle tissue under the electron microscope revealed loss of myofilaments and enlarged, irregular mitochondria embedded in sarcoplasmic areas filled with a particulate structure thought to be glycogen.

This patient did not respond to anabolic corticosteroid therapy, but clinical and electromyographic improvement occurred when prednisone was substituted for dexamethasone and the dosage of corticosteroid was slowly reduced.

Case 2

A man aged 38 years had suffered from ankylosing spondylitis for 13 years. After receiving dexamethasone, 2 mg. daily, for 19 months he complained of loss of weight in addition to stiffness and twitching of the leg muscles. Voltage–duration curves were normal, but the EMG showed a myopathic pattern (Plate V, Fig. 2). Serum creatine was 0·3 mg. per 100 ml. (normal, 0·2 to 0·7 mg.); serum creatinine 0·6 mg. per 100 ml. (normal, 0·9 to 1·7 mg.). Serum aldolase was 1·8 Bruns units and S.G.O.T. 9 sigma Frankel units. ECG normal.

Muscle biopsy showed an excess of central nuclei with a tendency to nuclear chain formation. Electron microscopy of muscle showed loss of myofilaments and disruption of the sarcoplasmic reticulum, in which large and apparently functionless mitochondria were dispersed.

The patient improved rapidly when the corticosteroid dosage was reduced, and there was commensurate improvement in the EMG.

Case 3

A man aged 44 had suffered from rheumatoid arthritis of three years' duration and had received dexamethasone 4 mg. daily for three months. He complained of pain in both thighs and weakness of the legs. Voltage–duration curves from both quadriceps were normal. EMG demonstrated a myopathic pattern. Serum creatine 0·2 mg. per 100 ml., and serum creatinine 0·7 mg. per 100 ml. Serum aldolase was raised to 19·6 Bruns units and S.G.O.T. was 10 sigma Frankel units. ECG normal.

Muscle biopsy showed small and scattered areas of phagocytosis and formation of short nuclear chains in a few places. Electron microscopy revealed some destruction of myofilaments.

This patient represented the mildest example of myopathy in this series. He showed rapid clinical improvement as the corticosteroid was withdrawn.

Case 4

A woman aged 48 had suffered from severe rheumatoid arthritis for nine years. She had received corticosteroid therapy for 3½ years, having initially taken cortisone 75 mg. daily for six months followed by triamcinolone 8 mg. daily for 11 months, dexamethasone 2 mg. daily for 12 months, and then triamcinolone 12 mg. daily for 12 months. There was gross muscle weakness and wasting of the quadriceps. Voltage–duration curves from the

right vastus medialis were normal apart from a high rheobase, but stimulation of the left vastus medialis failed to produce a response with voltages up to 80. EMGs demonstrated a myopathic pattern. Serum creatine was 1·7 mg. and serum creatinine 0·7 mg. per 100 ml. Serum aldolase 6·1 Bruns units, and S.G.O.T. 9 sigma Frankel units. ECG normal.

Muscle biopsy showed a few small areas of phagocytosis in addition to nuclear proliferation and clumping with some formation of short chains of dark nuclei. Electron microscopy showed loss of myofilaments.

Case 5

A man aged 52 had suffered from rheumatoid arthritis for seven years in addition to hypertension and myocardial ischaemia. He had received prednisolone 10 mg. daily for 15 months. Progressive weakness and wasting of the proximal muscles of both upper and lower limbs developed. Voltage–duration curves were attempted on both quadriceps and both deltoids, but no response was obtained with voltages up to 80. Serial EMGs demonstrated progressive myopathic changes. Urinary 24-hour creatine output was 70 mg. and creatinine 1,177 mg. Serum aldolase was 5·5 Bruns units. Muscle biopsy showed gross atrophy of muscle fibres with loss of cross-striations, areas of phagocytosis and muscle necrosis, proliferation of dark-staining nuclei in clumps, and infiltration of the muscles by adipose tissue. Electron microscopy showed severe loss of myofilaments, with enlarged and probably functionless mitochondria.

The patient died suddenly, and autopsy established that death was due to acute myocardial insufficiency resulting from severe coronary atherosclerosis, coincident with rheumatoid arthritis and atherosclerosis of larger limb arteries.

Histologically, many of the smaller arteries showed panarteritis with fibrinoid necroses of media and intima associated with diffuse infiltration by lymphocytes, plasma cells, and occasional eosinophils (Plate V, Fig. 3). These small vascular lesions were demonstrated in limb muscles, vasa vasorum of larger arteries, in and around peripheral nerves, and in the subserosa of the small intestine, but not in other viscera. The lesions in voluntary muscle resembled those seen in the biopsy, endomysial fatty infiltration being the most prominent feature.

The degeneration of voluntary muscle fibres and arteritic lesions in voluntary muscle occurred irregularly. Severe fatty degeneration was observed in several areas of striated muscle supplied by vessels of normal appearance, suggesting that the muscular lesions were not solely dependent upon vascular disease (Plate V, Fig. 4).

Case 6

A man aged 51 who had suffered from rheumatoid arthritis for eight years and who had never received any corticosteroid therapy. He complained of weakness and loss of weight (nearly 28 lb. in six months), and had generalized wasting, particularly involving the thigh muscles. Voltage–duration curves were normal. EMG demonstrated a myopathic pattern. Serum creatine 0·7 mg. and creatinine 0·6 mg. per 100 ml. Serum aldolase raised to 12 Bruns units. S.G.O.T. normal at 9 sigma Frankel units. Muscle biopsy showed some nuclear proliferation. ECG normal.

Case 7

A woman aged 73 had suffered from rheumatoid arthritis for four years. This patient had lost over 40 lb. in weight in the past year, but had no symptoms apart from muscular weakness and the pains and stiffness due to her arthritis. Investigations, including radio-

graphy of chest and barium-meal examination, did not reveal any evidence of other disease. ECG normal. Serum creatine 0·6 mg. and serum creatinine 0·8 mg. per 100 ml. Serum aldolase 9·3 Bruns units and S.G.O.T. 8 sigma Frankel units. Muscle biopsy showed some nuclear proliferation and clumping in otherwise normal fibres.

DISCUSSION

There is considerable evidence that myopathy may be induced by excess of corticosteroid hormone, irrespective of whether that excess results from disease (Cushing's syndrome), from administration of hormones to experimental animals, or during the treatment of various diseases by cortisone and its synthetic analogues. The frequent occurrence of myopathy in patients receiving corticosteroid therapy for allergic, haematological, and dermatological conditions indicates the exclusive role of corticosteroids in causing muscle lesions in these patients. With regard to patients receiving anti-inflammatory corticosteroids in the treatment of rheumatic disorders, particularly rheumatoid arthritis, the situation is complicated by the fact that these conditions can themselves cause a myopathy, and we have briefly described two such patients (Cases 6 and 7) in whom the myopathic changes were severe.

Muscle lesions complicating rheumatoid arthritis may be due to disuse atrophy consequent upon prolonged immobilization; atrophy in muscles acting upon inflamed joints; diffuse myositic lesions ranging from scattered patches of round-cell infiltration (lymphorrhages) to severe polymyositis; ischaemic changes in muscle secondary to arteritis, and even areas of focal infarction; and muscular changes consequent upon neuropathy which may be associated with arteritis of the vessels supplying the nerves. Case 5 illustrates the difficulties encountered in differentiating between a muscle disorder associated with the rheumatoid process and a myopathy due to corticosteroid therapy. This patient did not show clinical evidence of any type of neuropathy, although arteritis was demonstrated in relation to some peripheral nerves, particularly the lateral popliteal nerve. It is unlikely that all the muscle changes were entirely due to ischaemia and necrosis consequent upon vascular insufficiency. The clinical presentation of increasing weakness and wasting of the proximal muscles of the limbs, in addition to progressive myopathic changes on serial electromyograms during the course of substantial dexamethasone therapy, suggested that the changes were at least partly due to the corticosteroid.

In Cases 1, 2, and 3 there has been retrospective proof of the association between the myopathy and corticosteroid therapy, since these patients improved when the dosage was reduced and the drug withdrawn. The numerous reports citing fluorinated corticosteroid preparations as a cause of myopathy has given some strength to the suggestion, as yet unproven, that the fluorine atom in these preparations may be culpable. It is therefore reasonable, as an initial measure in

management, to change to a non-halogenated compound—e.g. prednisone, prednisolone, or methyl-prednisolone—and then to reduce the dosage gradually. Treatment with an anabolic corticosteroid did not appear to confer any benefit in Case 1, while the patient was still receiving a substantial dosage of corticosteroid, and in Case 4 there has been no response to anabolic corticosteroid therapy given while the patient has continued to take 8 mg. of triamcinolone daily.

Conventional histological examination has revealed only minor changes in all cases, with the exception of Case 5. Despite gross weakness and wasting clinically, there has been no evidence of extensive fibre necrosis or atrophy. Inflammatory signs have been scanty, amounting to no more than occasional small areas of phagocytosis. The occurrence of a moderately increased number of dark-staining sarcolemmal nuclei and occasional nuclear clumping and short-chain formation are the most characteristic features, as these findings were noted in all the specimens of muscle examined.

Electron microscopy has shown more striking changes, and has also indicated several characteristics common to the specimens examined from Cases 1 to 5, at microscope magnifications ranging from 14,000 to 45,000. There is loss of myofilaments with fragmentation; mitochondria are often enlarged and probably functionless, and are seen in the sarcoplasmic areas between the broken myofibrils. The sarcoplasmic reticulum is also disrupted. Some normal mitochondria and sarcoplasmic reticulum may also be seen adjacent to relatively intact myofibrils. Further studies on these specimens will be reported in detail later. Investigations of other types of myopathy are essential for purposes of comparison in order to determine the existence of features specific to a given type.

SUMMARY

The clinical, electromyographic, and histological appearances of myopathy in five patients who had received corticosteroid therapy are described. The case-histories of two patients suffering from myopathy apparently complicating rheumatoid arthritis have also been presented.

Conventional histological methods revealed only minor changes in the muscle fibres in four patients, the significant findings being a proliferation of darkly stained sarcolemmal nuclei with clumping and short-chain formation. The fifth patient showed gross muscle necrosis with fatty infiltration, and at autopsy (following death from acute myocardial insufficiency) a diffuse rheumatoid arteritis was demonstrated.

Specimens of muscles from these patients have been examined under the electron microscope, and the principal features noted were loss of myofilaments with fragmentation of their ends; large and apparently functionless mitochondria in the sarcoplasmic areas between the broken myofilaments; and a particulate structure, thought to be glycogen, in the disrupted sarcoplasm.

Acknowledgments

We wish to thank Professor A. G. Hepplestone and Dr. J. N. Walton for granting facilities for this work and for their advice. The technical assistance given by Mr. Hardy and Miss Jenkinson is also acknowledged. The expenses involved in the electron microscopy in this work were met by a grant from the Canadian Muscular Dystrophy Fund.

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ORIGINAL PAPER

THE NEWER CORTICOSTEROIDS FOR LOCAL INJECTION*

BY G. HOLDEN and P. HUME KENDALL

From the Department of Physical Medicine, Guy's Hospital, London

ALTHOUGH local corticosteroid injection therapy has now found a well-established place in the treatment of skeleto-muscular disorders, the perfect preparation for soft-tissue and synovial injection has yet to be developed. A great deal of time was devoted to the study of the various homologues of hydrocortisone following Hollander's first report in 1951, but there seemed to be little or no practical difference between them. Prednisolone appeared to show some superiority over hydrocortisone and has been the subject of several favourable reports; however, many of the undesirable features of cortisone are evident in prednisolone, which justifies the continued search for the ideal corticosteroid. Recently several more potent derivatives have become available which offer advantages over the older corticosteroids when administered orally. Theoretically, these should have a comparable effect when used by local injection. It is in the pursuit of the ideal corticosteroid that this report on three new preparations—namely, triamcinolone diacetate, triamcinolone acetonide, and 6-methyl prednisolone (Medrone)—is presented (see Table I).

TABLE I

NEWER CORTICOSTEROIDS FOR INTRA-ARTICULAR INJECTION

Preparation	Concentration in Suspension (mg. per ml.)
Triamcinolone diacetate	20
Triamcinolone acetonide	10
6-Methyl prednisolone	40

Several desirable features are required of the ideal corticosteroid. These are: (1) a long duration of activity; (2) little or no post-injection pain; and (3) no demonstrable side-effects. So far these have not always been allied to clinical effectiveness in the corticosteroids used for local injection. It therefore seemed important to evaluate these factors in the new products. Clinical trials were planned to assess duration of action as compared with other standard corticosteroids, and also to test their potency in conditions that have proved resistant to other injections.

* Paper read at the Annual Meeting of the British Association of Physical Medicine, London, April, 1961.

TRIAMCINOLONE DIACETATE AND ACETONIDE

Initial reports of the use of suspensions of triamcinolone diacetate and acetonide suggested that these drugs had appreciable anti-inflammatory activity when used by local injection. Dordick and Bernstein (1959) claimed that "excellent to satisfactory responses, as measured by the decreased joint pain and stiffness", followed injection of triamcinolone in 32 out of 36 cases. The duration of effective joint response was from four to six weeks. Sperling (1960) also stated that "a greater remission of joint symptoms was achieved with triamcinolone than with other forms of intra-articular steroids".

Our experience has not confirmed these initial reports. A series of 49 intra-articular injections were given into the joint of patients with rheumatoid arthritis, who had either previously received or were subsequently given injections of prednisolone acetate. The effects of the injection on joint pain, range of movement, and swelling were studied, and the patient was asked to report on the presence or absence of post-injection pain. The mean duration of action after injection of triamcinolone was 4·7 days, and after prednisolone 8·7 days (Table II).

TABLE II

COMPARISON OF THE ACTION OF PREDNISOLONE AND TRIAMCINOLONE WHEN GIVEN BY LOCAL INJECTION

	No. of Patients	Duration of Action (Days)
Prednisolone	73	8·7
Triamcinolone	49	4·7

Thirty-four injections were given to patients suffering from soft-tissue lesions. The success rate was 29—that is, 82%. Ten injections were given in resistant cases; only one of these was successful.

Post-injection pain was noted in 7 out of the total of 93 injections—that is, 8%. There seems little doubt that the triamcinolone preparations are effective when used for local injection, but they appear to hold out no advantage over the existing corticosteroids available.

MEDRONE

The main part of the investigation concerned the use of 6-methyl prednisolone. This comprised the study of the results of injection in a variety of cases of soft-tissue and arthritic lesions. The success rate, duration of relief from pain, and duration of post-injection pain were also estimated.

The total number of cases injected was 103, including 53 cases of rheumatoid arthritis, 13 of osteoarthritis, 23 of acute tendinous and ligamentous lesions, and

14 of "rotator-cuff syndrome". All these conditions would normally be expected to show some response to local corticosteroid injections. The results of joint injection in these patients are shown in Tables III and IV.

TABLE III

RESULTS OF INJECTING VARIOUS SOFT-TISSUE LESIONS WITH 6-METHYL PREDNISOLONE

Clinical Group	No. of Cases	Duration of Injection Pain (Hours)	No. of Cases Improved	No. of Cases Failed
Rotator cuff syndrome	14	26·4	13	1
Tendon and ligamentous lesions . .	23	33·6	22	1

TABLE IV

RESULTS OF INJECTING ARTHRITIC LESIONS WITH 6-METHYL PREDNISOLONE AND PREDNISOLONE

Clinical Diagnosis	Average Duration of Relief of Pain (Days)	
	With 6-Methyl Prednisolone	With Prednisolone
Rheumatoid arthritis (53 cases)	13·2	8·7
Osteoarthritis (13 cases)	8·7	4·6

The duration of post-injection pain is very little different from the average obtained with prednisolone. The success rate is, however, quite high, and is better than one would expect with prednisolone. The results of injection of rheumatoid arthritic joint lesions reveal a clear advantage over prednisolone. The average duration of relief from pain was 13·2 days as compared with 8·7 days with prednisolone. A similar improvement was shown in osteoarthritis. These comparisons were made by injecting the affected joint with each corticosteroid separately, or, if this proved impossible, a similarly inflamed joint in a similar patient was used as the control. It was interesting to note that the marked increase of pain in arthritic cases was not observed in the present series when using 6-methyl prednisolone.

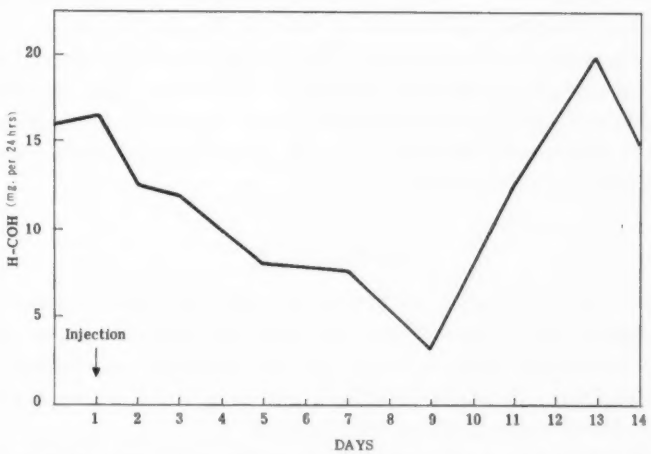
A further study was designed to ascertain whether 6-methyl prednisolone would prove effective when resistance had already been demonstrated to two or more previous injections of other prednisolone salts. Thirty-two resistant soft-tissue

lesions received a single injection of Medrone with a subsequent success rate of 87%; in only four cases was there failure to respond.

SYSTEMIC ABSORPTION

Several patients with rheumatoid arthritis noticed a general improvement in other joint symptoms, which usually lasted for one to two days after the injection. It seemed possible that this was the result of systemic absorption. In order to investigate this possibility two patients were given an injection of 40 mg. of 6-methyl prednisolone (the equivalent to 50 mg. of prednisolone acetate). The daily total output of urinary 17-hydroxycorticosteroids was estimated by the method of Appleby *et al.* (1955) and the results plotted as a graph (see Figure). This showed that there is an initial depression from the resting level preceding injection. The fall continued until the ninth day and then rose abruptly, to return to resting levels after 12 to 14 days.

These results suggest that there is almost certainly an appreciable systemic absorption of the corticosteroid over a period of several days.



Graph showing total 17-hydroxycorticosteroid excretion after a single intra-articular injection of 6-methyl prednisolone, 40 mg.

DISCUSSION

There appeared to be a considerable variation in the properties of the two groups of corticosteroids investigated when used by local injection; whereas triamcinolone is certainly no more active, and possibly less effective, than either prednisolone or hydrocortisone, 6-methyl prednisolone has an appreciably longer duration of action, appears to possess a more potent anti-inflammatory action,

and showed a high degree of activity when other corticosteroids had failed to relieve symptoms.

It is difficult to understand how this discrepancy between two similar corticosteroids can occur when they are given by local injection, whereas when given orally there is an almost identical clinical effect. The difference in activity of any corticosteroid when injected locally appears to be a product of: (1) basic anti-inflammatory properties; rate of breakdown at the site of action; and (3) rate of absorption from the joint. All three of these factors may differ with each new corticosteroid, which possibly accounts for some of the variation.

The whole question of systemic absorption following local injection of a corticosteroid is becoming increasingly more important and complex. The apparent suppression of adrenal function that appears to follow a single injection of 40 mg. of 6-methyl prednisolone may, in fact, be an incorrect interpretation. Greaves and West (1960) have pointed out that there is considerable evidence that corticosteroids act as catalysts and that their metabolites result largely from the disposal mechanisms in the liver; as a result there may be some disparity between the quantities of circulating free hydrocortisone and the excretion of 17-hydroxy-corticosteroid metabolites. Thus it seems questionable whether there is in fact a true depression of suprarenal activity or whether the suppression of 17-hydroxy-corticosteroids presents a false picture. This remains to be elucidated. Certainly as yet no clinical evidence of adrenal suppression following local corticosteroid injection has been recorded, but the possibility must constantly be borne in mind as more potent corticosteroids become available, particularly in old people who have a less labile endocrine metabolism.

SUMMARY

Clinical evaluation of two new groups of corticosteroid preparations for local injection suggests that triamcinolone acetonide and diacetate show no material advantage over existing corticosteroids, but that 6-methyl prednisolone possesses a significantly longer duration of activity and is effective where other corticosteroids have failed to bring about improvement.

Initial investigations suggest that there is appreciable systemic absorption of 6-methyl prednisolone and possibly some depression of adrenal activity. The significance of this is discussed.

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FIG. 1.—Photograph showing the swelling.

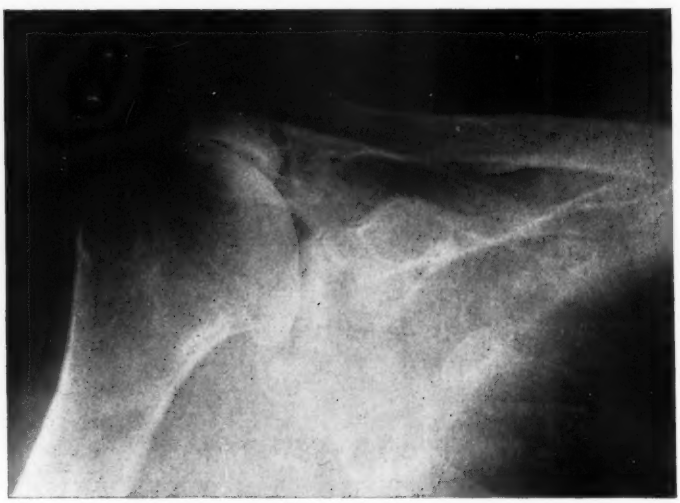

FIG. 2.—Radiographic appearances.

CLINICAL REPORT

SYNOVIAL CYST OF THE ACROMIO-CLAVICULAR JOINT

By J. M. EVANS

From the Middlesex Hospital, London

MORRANT BAKER was the first to describe synovial cysts of the knees in 1877. These were usually associated with osteoarthritis and sometimes precipitated by trauma. The synovial membrane was thought to have herniated through a weakened posterior capsule. Similar cysts in other joints must be rare. A patient has recently been seen with a synovial cyst at the acromio-clavicular joint, and a review of the literature failed to reveal another case, although Berbrayer (1958) has described one case of cystic swelling of the intra-articular disk of the acromio-clavicular joint. The following case history may therefore be of interest.

CASE HISTORY

The patient, a 76-year-old widow, gave a six-year history of pain in her shoulders, knees, and lumbar spine due to osteoarthritis. Six months before attending hospital she had fallen on to the right shoulder, and a progressive swelling had developed over the right acromio-clavicular joint, causing sufficient pain to wake her at night. Right shoulder movement had become limited, particularly on abduction.

Examination showed a slightly fluctuant tense swelling, about 2 × 1½ inches, over and firmly attached to the right acromio-clavicular joint (Plate VI, Fig. 1). This was not tender, red, or hot, and could be transilluminated. Abduction at the right shoulder was painful and limited to 160 degrees, and there was slight limitation of external rotation. Her general condition was good for her age, and there were no abnormal signs in the nervous system or in the joints, apart from Heberden's nodes. Radiographs of the right shoulder showed gross deformity of the humeral head with cystic changes and marginal lipping. The adjacent margins of the clavicle and acromion were flattened and deformed, and there was a soft-tissue swelling above the joint (Plate VI, Fig. 2). These appearances suggested advanced osteoarthritis. Blood count, erythrocyte sedimentation rate, and serum uric acid level were normal. Rose's test, Wassermann reaction, and Price precipitin reaction were negative. X-ray appearances of the chest were normal.

Aspiration of the swelling produced 10 ml. of clear, yellow, mucous fluid which clotted on standing and contained numerous red and white blood cells, with 5% polymorphs and 95% lymphocytes; the protein content was 3 g. per 100 ml. Nothing was grown on culture, although a search was made for tubercle bacilli. The fluid rapidly reaccumulated after removal, and the patient was therefore referred to the orthopaedic department, where the cyst was excised under local anaesthesia by Mr. P. H. Newman. He reported that the cyst had been multiloculated, full of rather darkly stained synovial fluid, and had to be scraped from the underlying bone to which it was adherent. There were obvious osteoarthritic changes in the underlying joint. These findings were consistent with the

diagnosis of a Morrant Baker cyst, but unfortunately no histological examination was made.

Excision of the cyst, however, has not relieved her pain, and removal of the end of the clavicle is being considered.

DISCUSSION

Previous radiographs in this case had shown osteoarthritis of the acromio-clavicular joint. Following a fall, and probably precipitated by it, a tense cyst developed over the joint. Rheumatoid arthritis, gout, a neuropathic joint, and an infective lesion were excluded. The cyst was eventually excised, both for curative and for diagnostic purposes, and was found to be identical to the cysts described by Morrant Baker as occurring in the knee-joint.

ACKNOWLEDGMENT

I am indebted to Mr. P. H. Newman for permission to publish this account.

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REVIEWS OF BOOKS

DIAGNOSIS IN LOCOMOTOR DISORDERS. By Kenneth Stone. *Pp. 224. 25s. London: Oxford University Press. 1960.*

This book consists of twenty-five chapters devoted to the differential diagnosis of symptoms and signs commonly encountered in disorders of the locomotor system. The chapters are arranged in alphabetical order, starting with Aching, Ataxy, and Atrophy, and concluding with Paraesthesia, Polyarthritis, and Spasticity.

This comprehensive book is well set out and fully illustrated, though some of the radiographs have reproduced badly. It will be useful to those who wish for a book of quick reference, particularly as, until now, there has been no book of this kind devoted to disorders of the locomotor system.

H. A. BURT

A PHYSICIAN'S INTRODUCTION TO ELECTRONICS: A LABORATORY MANUAL. By A. C. Morris, Jr. *Pp. 43. 15s. London: Pergamon Press. 1961.*

The subtitle of this book indicates its aim, which is to provide a series of exercises using the Orins electronic breadboard to study the behaviour of capacitors, rectifiers, and valves.

There is an introductory seven pages giving definitions of the terms commonly used in electronics. The explanations of such terms as "reactance" and "impedance" are too abbreviated to be helpful to the tyro, and no attempt is made to explain valve theory or time bases. In short, the book has no value unless used in connexion with a course of lectures and demonstrations and must therefore have a very limited appeal to physical medicine specialists in Great Britain.

C. B. WYNN PARRY

LES TRACTIONS VERTÉBRALES. By J. Levernieux. *Pp. 124. No price. Paris: Expansion Scientifique Française. 1960.*

This small monograph on spinal traction can easily be read by anyone with a modest knowledge of the French language. Methods of traction are illustrated and the results of treatment of several thousand patients are set out in tables. The ill effects of treatment are described in a short section at the end of the book, though these were fortunately rare. I recommend this book to those interested in the treatment of pain in the neck and back.

S. MATTINGLY

LUMBAR DISCOGRAPHY AND LOW BACK PAIN. By Donald deForest Bauer. *Pp. 89. 36s. Springfield, Illinois: Thomas. 1961.*

In this monograph Dr. Bauer describes the technique and applications of discography. Briefly the technique consists in the injection of a radio-opaque substance into the nucleus of a disk, thereby giving information which is not obtainable by straight radiography or myelography. The author claims that the procedure is not dangerous, although somewhat more painful than myelography.

The technique of discography is not new in the U.S.A. or Europe, but is not a popular procedure in the United Kingdom.

The book is well produced and the reproduction of the many radiographs of a high standard.

B. G. B. CHRISTIE

PARENTS OF THE HANDICAPPED. By Alfred H. Katz. *Pp. 155. 48s. Springfield, Illinois: Thomas. 1961.*

This book is not about the parents of handicapped children, but about some of the groups into which such parents have formed themselves in the United States of America. It is a study of what Gunnar Dybwad, in the foreword, refers to as "this new social phenomenon", "the sudden and forceful emergence of self-organized groups of parents (and friends and relatives) of the handicapped".

The book is concerned chiefly with the development of four large parents' associations centred on New York and their relationships to their own members, but their relationships to established welfare organizations and to the medical profession are also considered. Readers in Great Britain—and doubtless in many other countries—will be struck by the similarities between the developments described and those with which they are familiar closer home.

WILLIAM DUNHAM

ABSTRACTS OF THE LITERATURE

Immunity Mechanisms in Neurological Disease. W. H. McMenemy. *Proc. roy. Soc. Med.*, 1961, **54**, 127.

This is a long, closely argued paper which formed the presidential address for 1960–1 at the Section of Neurology of the Royal Society of Medicine.

The relation of protein pattern in blood on electrophoresis to that of cerebrospinal fluid is described. Recent theories on autoimmunity as a cause of such disorders as encephalitis and disseminated sclerosis are critically discussed with special reference to Burnet's hypothesis of clonal selection. Although these are speculative only at the present state of knowledge, they provoke thought and stimulate ideas.

[This paper is well worth close study by all who manage neurological disorders.]

C. B. Wynn Parry

Reversible Block of Nerve Conduction by Ultrasound. R. R. Young and E. Henneman. *Arch. Neurol. (Chicago)*, 1961, **4**, 83.

The authors studied the effect of focused ultrasound on axonal conduction in a mixed nerve in frogs. They found that conduction can be blocked reversibly, and irreversibly in higher dosage. The small fibres are most sensitive and can be differentially blocked without affecting the larger fibres.

C. B. Wynn Parry

A Study of Abnormal Reflex Patterns in Spasticity. T. C. Hohmann and J. Goodgold. *Amer J. phys. Med.*, 1961, **40**, 52.

This paper describes an investigation into the presence of the H reflex in normal subjects and patients with upper motor neurone lesions. The H reflex is a monosynaptic reflex recordable from quadriceps and calf muscles on stimulation of the motor nerve. It was infrequently found in normal subjects, but present in 25% of patients with upper motor neurone lesions. It has been suggested before that its presence in these disorders may be due to release of inhibitory pathways.

The authors propose the study of the H reflex as an additional electrodiagnostic tool in equivocal cases of upper motor neurone disorders.

C. B. Wynn Parry

Histology of the Neuromuscular Junction in Dystrophia Myotonica. V. MacDermot. *Brain*, 1961, **84**, 75.

Ten cases of dystrophia myotonica were subjected to biopsy and electromyography. For biopsy examination specimens were stained with methylene blue to show the histology of the nerve fibres and end-plates. In all cases abnormalities of the axons and myelin were present both in the distal nerve fibres and in the intramuscular nerve bundles. Abnormal tortuosity of nerve fibres was common and almost all the end-plates were abnormally small. The most striking abnormality was the extremely profuse terminal branching of the nerve fibres with the formation of multiple end-plates on the same muscle fibre. All the patients showed high-frequency discharges on electromyography and a myopathic pattern. The evidence suggests that in addition to a muscle-fibre abnormality there is also a neuronal defect in this disease.

C. B. Wynn Parry

A Review of Skeletal Muscle. A Review of Sensory Receptors in Skeletal Muscle. A Review of the Motor Unit. V. Harrison. *Phys. Ther. Rev.*, 1961, **41**, 17, 25, 30.

These are three reviews of modern knowledge about the structure of muscle, the spindle system, and the anatomy and electromyography of the motor unit. Each review is clear, concise, up-to-date, and fully documented. They are strongly recommended to all who are interested in electrodiagnosis.

C. B. Wynn Parry

The Neurology of Language. W. R. Brain. *Proc. roy. Soc. Med.*, 1961, **54**, 433.

This is the Hughlings Jackson lecture for 1961. Those who heard it will now have an opportunity to study closely the exciting new ideas and concepts of the anatomy, physiology, and psychology of language and their relation to aphasia. For those who did not hear the lecture this paper is a "must". It will clarify many obscurities and be of value in the management of aphasic patients, as well as opening up a whole new field of ideas. The paper is too closely argued and full of challenging and stimulating concepts for easy abstraction. The only advice that can be given is—"Read it".

C. B. Wynn Parry

Discussion on Normal and Abnormal Willed Movement. A. J. Buller and O. C. J. Lippold. *Proc. roy. Soc. Med.*, 1961, **54**, 199.

Dr. Buller describes the phasic and static stretch reflexes, relating the former to the gamma system and the latter to the Renshaw loop. He discusses the meaning of tone and some of the unsolved problems of muscular control.

Dr. Lippold discusses tremor and concludes that the superimposed rhythmical tendency in normal voluntary muscular activity is due to activity in the reflex arc.

[Both these papers are beautifully clear and of great importance to specialists in physical medicine.]

C. B. Wynn Parry

Medial Genicular Fat Pad Thrombosis as a Cause of Knee Pain. I. M. SIEGEL and S. SIDEMAN. *J. Amer. med. Ass.*, 1961, **176**, 691.

Fat pads on the medial side of the knee-joints are commonly seen in middle-aged obese females, and in many cases these pads strike each other during ambulation. In the authors' view this repeated trauma leads to venous thrombosis in the fat pads, and such a case is described; the segment of thrombosed vein could be palpated superficially in the fat pad. Treatment consists of rest and supportive measures, and X-ray therapy may help. Weight reduction, correction of varicosities, and gait training may prevent recurrences.

B. E. MACE

Quadriceps Myopathy Occurring in Middle Age. J. W. A. TURNER and K. W. G. HATHFIELD. *J. Neurol. Neurosurg. Psychiat.*, 1961, **24**, 18.

This is a report of two cases, both in women aged 43 and 52 respectively, the former being under observation since 1939 (Denny-Brown, *Proc. roy. Soc. Med.* (Sect. Neurol.), 1939, **16**, 1), and only gradual spread of weakness has occurred.

Details of muscle biopsy are given and the electromyogram is described. The former suggests the condition is a chronic polymyositis rather than progressive muscular dystrophy.

MAURICE F. HART

Osteoarthritis. W. J. TOBIN. *J. chron. Dis.*, 1961, **13**, 495.

This is a review of the more important underlying gross pathological changes in osteoarthritis and spondylosis. Aetiology is dealt with in general terms, but the part played by trauma in causing osteoarthritis and in aggravating pre-existing disease is discussed in some detail, with reference to medico-legal problems arising in such cases from compensation claims. General principles of management are reviewed and surgical procedures are briefly mentioned in connexion with the hip, knee, foot, and localized lesions of the lumbar spine.

CHARLOTTE FELDMAN

Current Methods of Treatment: Osteoarthritis of the Spine. R. L. PRESTON. *Arthr. and Rheum.*, 1961, **4**, 213.

The author confines his study to the lumbar spine, the mechanics of which he discusses fully, pointing out that the pain caused by soft-tissue inflammation is reversible; bone and cartilage changes are not.

Most cases respond well to adequate treatment. In severe pain, rest till muscle relaxation occurs is essential. Mild heat and massage may be given at this stage. Later extension exercises are begun and should be continued for years. A survey of the patient's daily activities is made and adjusted where necessary. Only rarely, in localized disease, are operative measures considered, and spinal supports are seldom needed.

MARGARET HOLROYDE

Carpal Tunnel Syndrome—Initial Manifestation of Systemic Disease. L. A. GRISSMAN, H. J. KAPLAN, F. D. OWNBY, and M. CROSSMAN. *J. Amer. med. Ass.*, 1961, **176**, 259.

Three cases with a primary complaint of nocturnal pain and paraesthesiae of the hand are described. All were found to have signs of median-nerve compression, which was relieved surgically. The predisposing diseases were rheumatoid arthritis, multiple myeloma, and gout, and in the first two cases the symptoms referred to the median neuropathy preceded all other manifestations of the disease.

B. E. MACE

Diagnosis and Treatment of Lupus Erythematosus, Dermatomyositis, and Scleroderma, with Emphasis on Cutaneous Findings. R. K. WINKELMANN. *J. chron. Dis.*, 1961, **13**, 401.

In this paper the author utilizes his wide experience in the Section of Dermatology at the Mayo Clinic to give a detailed account of the cutaneous manifestations of lupus erythematosus and related conditions, dermatomyositis and scleroderma. Other clinical features and laboratory findings of diagnostic importance are discussed, while the treatment, which is different in each of these three conditions, is concisely and clearly summarized.

CHARLOTTE FELDMAN

Sunlight and the Skin. J. M. KNOX. *J. chron. Dis.*, 1961, **13**, 391.

This paper provides fundamental information related to the skin and sunlight, with stress on the damage that can result from exposure to the latter. Sunburn and sun-tan, actinic degeneration, and carcinogenesis are dealt with. Photosensitivity—the result of individual allergy, administration of certain drugs, as a cutaneous manifestation of systemic disease, or as a rare familial condition (keratoderma pigmentosa)—is discussed.

In the prevention and management of harmful effects of sunlight the author considers the importance of detailed advice to the patient and the topical use of substances which act either by scattering light or by absorbing specific wavelengths. The literature dealing with the mechanism of action of the antimalarial drugs in lupus erythematosus and light-sensitive diseases is also reviewed briefly.

CHARLOTTE FELDMAN

Modern
deep-heating
techniques
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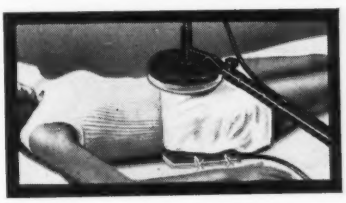

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